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VARIATIONS IN NORMAL GASTRIC FUNCTIONS AND THEIR CAUSES: SOME NEW EXPERIMENTAL AND CLINICAL OBSERVATIONS.[1]

By FRANK L. APPERLY, M.A., M.D. (Oxon), D.Sc. (Melb.),
Honorary Physician to Out-patients, Saint Vincent's Hospital, Melbourne; Senior Lecturer in Pathology, University of Melbourne;

AND

KATHLEEN M. SEMMENS, B.Sc. (Melb.)
[From the Macleod Laboratory, Pathology Department, University of Melbourne.]

SECTION I: THE STUDY OF GASTRIC PHYSIOLOGY AND PATHOLOGY BY MEANS OF THE FRACTIONAL TEST MEAL.

THE study of normal variations of bodily function has not up to the present received the attention it deserves. Yet these variations and their causes are of immense value in that they give us the clue to the causes of abnormal variations, the beginning of pathology and medicine. Physiology has still much to do for medicine.

In this paper are recorded our experiments and observations on normal variations of gastric function, using the fractional test meal as our principal method.

Difficulties in the Interpretation of the Fractional Test Meal.

The fractional method up to the present time has failed to be of the practical value at first anticipated. At first enthusiasts believed that each clinical variety of gastric dysfunction would be represented by a definite type of acidity curve and that the method would present us with a ready-made diagnosis whenever it was used. These expectations were not realized and, even in expert hands, it has been found impossible in the majority of cases to interpret the various curves on a test meal chart. Many would have it that the method is of no use except as a help in such conditions as gastric carcinoma, duodenal ulcer, pyloric stenosis and pernicious anæmia.

The reason for this attitude is that we have not sufficient knowledge to interpret these curves. In this paper we attempt to throw further light on the problem, so that test meal charts may be used to give an accurate account under the conditions of test of both chemical and muscular functions of the stomach and upper intestine over a period of time. Much of the work published so far has been apparently contradictory. One example will suffice. Early rapid emptying of the stomach and pyloric stenosis will each produce a hyperchlorhydria. But in the former instance it is an early hyperchlorhydria and in the latter a late hyperchlorhydria of the slow "climbing" type. This confusion is due to the facts that a number of different factors contribute to the make-up of the acidity and total chloride values at any moment and, secondly, that we have not known the effects of variations of each of these factors on the values mentioned.

[1] Much of the expense of this work has been met by the Edward Wilson (*Argus*) Trust.

Theoretical.

We know that when a fluid meal is placed in the stomach, several things occur before the latter empties. Suppose a volume a of fluid food enters the stomach. After the lapse of a period of time, say, one hour, a volume b of gastric juice of acid concentration B and sodium chloride concentration S has been secreted into this and a volume c of average acid concentration C has left the stomach to be partly replaced by fluid regurgitated from the duodenum of volume d and sodium chloride concentration E. At any moment the various values estimated in the gastric samples will be roughly as follows, assuming B and S to remain constant.

1. Total acidity (T.A.) $= \dfrac{bB - cC}{a + b - c + d}$

2. Total chloride (T.C.) $= \dfrac{bB + bS - cC + dE}{a + b - c + d}$

3. Neutral chloride (NaCl) $= \dfrac{bS + dE}{a + b - c + d}$

From inspection of a test meal chart, with curves representing total chloride, total acidity and neutral chloride, who can tell which of the factors, a, b, c, d, B, C, E or S has varied or been most responsible for variation of the final result from the normal average? Until we can, to some extent at least, do this and know something of the causes of these variations, we will never be able to read these charts.

In the work to follow we are chiefly concerned with variations in different individuals of the values of the acidity and total chlorides of the test meal up to one or one and a quarter hours. During this time the regurgitation of neutralized fluids from the duodenum to the stomach with dilution of gastric acid there is negligible, whatever may occur after this time. McLean's recent work[(1)] seems to show in fact that regurgitation plays little part in diluting gastric fluids at any time and that the fall of the acidity curve after one to one and a quarter hours is due to a diminution of the ratio of acid to neutral chlorides secreted in the gastric juice. (Incidentally, if the suggestion were put forward that this fall of acid production depended upon the alkaline tide in the blood, support would be given to such suggestion by our experiments on the effect of acidosis and alkalosis on gastric acidity to be detailed in the third subsection of Section 3.)

The effects produced by variations in single factors may be found by a consideration of the formulæ above and by comparing the theoretical results with those found by experiment and clinical observation. The effects of variation of bB (secretion of acid), of bS (the output of neutral chloride from the gastric mucosa) and of E (the concentration of neutral chloride in the duodenal fluids) are obvious from the formulæ. Variations of c are difficult to prove mathematically, but it is again obvious that with a rising acidity, the greater the loss of fluid from the stomach before the maximum acidity is reached, the less will be the dilution of the secreted acid by the gastric contents. In other words, the acidity will be raised by increase of c.

Formula 3 shows that rise of *c* also raises the neutral chloride. It follows therefore that the total chloride curve is raised also. A consideration of the results of gastro-enterostomy as in Figure II shows that increase of *d* lowers acidity, but has no effect on total chlorides.

The results on the test meal curves of variations in the various factors enumerated may be summarized as in Table I.

TABLE I.

SHOWING THE EFFECTS OF VARIATIONS OF VOLUME AND OF SALT, ACID OR ALKALI CONCENTRATIONS OF THE VARIOUS GASTRO-DUODENAL SECRETIONS ON THE CONCENTRATIONS OF TOTAL CHLORIDE AND ACID IN THE GASTRIC CONTENTS DURING A MEAL.

Rise in the value of	Effect on Test Meal Values of		
	T.C.	T.A.	NaCl.
bB (secretion of gastric acid)	+	+	0
bS (gastric secretion of neutral chloride)	+	0	+
c (rate of gastric emptying)	+	+	+
E (NaCl concentration of duodenal fluids)	+	0	+
D (alkali concentration of duodenal fluids)	0	−	+
d (volume of regurgitated duodenal fluids)	0	−	+

\+ sign indicates a rise of value.
− sign indicates a fall of value.
0 sign indicates no change.

As many of these factors vary in different individual charts, there can be only one way of proving these theoretical deductions and that is by classifying a large number of complete charts according as they vary in regard to one known factor, plotting the average of each class and comparing the changes produced. In the results that follow, all cases of achlorhydria have been excluded, as they appear to be a law unto themselves. The results obtained can then be compared with those shown in Table I.

SECTION 2: OBSERVATIONS ON THE RELATION OF VARIOUS GASTRIC FUNCTIONS TO ONE ANOTHER.

In the method adopted it has not been possible to study variations in some of the factors involved, for example, the volume and acidity of pure gastric juice, for reasons which will later be obvious. The effects of other variations are as follows:

Emptying Time of the Stomach.

Table II shows the average total chloride, total acidity and inorganic chloride in: (i) forty-one healthy young men, mostly students, and a few ex-hospital patients who had suffered from various surgical conditions of the limbs; (ii) twenty-five patients suffering from various non-organic dyspepsias; (iii) the averages of the above two classes combined. These have all been classified according to their emptying time as judged by the disappearance of the starch-iodine reaction in the samples drawn. The results in the combined group (iii) are shown in Figure I. Those with achlorhydria were omitted.

These results show that a short emptying time is associated with a lowered acidity curve and an earlier fall, while the total chloride curve is unaffected. McLean's work,[1] already referred to,

TABLE II.

Group and Emptying Time.	Number of Cases.	Observation made.	Values at Hours. ½ hour.	¾ hour.	1 hour.	1¼ hour.
Group I. (41 normal men.)						
0–1 hour	4	T.C.		66	78	78
		T.A.	17	30	34	26
		NaCl.		36	44	52
1–1½ hours	16	T.C.		58	71	80
		T.A.	22	32	38	40
		NaCl.		26	33	40
1½–2+ hours	21	T.C.		59	73	80
		T.A.	24	35	41	43
		NaCl.		24	32	37
Group II. (25 gastric patients.)						
0–1 hour	3	T.C.	56	76	73	90
		T.A.	29	35	23	30
		NaCl.	27	41	50	60
1–1½ hours	11	T.C.	57	73	81	89
		T.A.	25	35	40	41
		NaCl.	32	38	41	48
1½–2+ hours	11	T.C.	58	73	75	92
		T.A.	36	46	51	61
		NaCl.	22	27	24	31
Groups I and II combined.						
0–1 hour	7	T.C.		70	76	83
		T.A.	22	32	30	28
		NaCl.		38	46	55
1–1½ hours	27	T.C.		64	75	84
		T.A.	23	34	38	41
		NaCl.		30	37	43
1½–2+ hours	32	T.C.		64	74	84
		T.A.	28	38	44	49
		NaCl.		26	30	35

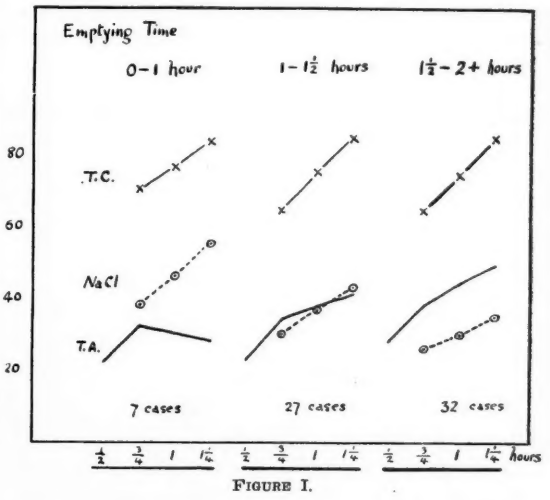

FIGURE I.

would suggest that in these rapidly emptying stomachs the proportion of acid to neutral chloride in the secreted gastric juice is diminished, that is to say, that there are wide variations among different individuals in the amount of acid secreted into a standard test meal. For reasons to be stated later in the next subsection but one, we do not believe that this is so. The differences between these groups

seem more to resemble the differences between the test meal charts before and after gastro-enterostomy and hemi-gastrectomy. In these we know that a diminished acidity curve with an unaffected total chloride curve and a more rapid evacuation are brought about by an increased dilution and neutralization of gastric contents following increased duodenal regurgitation. On these grounds we therefore believe that early emptying of the stomach is due to the same phenomenon. All of this implies a greater and more free secretion of pancreatic alkali. As will be shown later, when we experimentally produced an artificial alkalosis with increased secretion of alkali by the kidney and probably by the pancreas as well, the stomach emptied much more rapidly, with a lowered acidity or even an achlorhydria (see the third subsection of Section 3).

It might of course be objected that the pylorus controls emptying time. This is true, but it has been shown by Apperly[2] that the pylorus is in its turn controlled by duodenal regurgitation which neutralizes and dilutes free acid in the upper duodenum, thus removing the acid control of the pylorus and indirectly controlling emptying time of the stomach.

Note that in those cases in Table II and Figure I in which the starch reaction has disappeared before one hour, there still nevertheless remains sufficient fluid up to one and a quarter hours to estimate total chlorides, acidity *et cetera,* probably due to a persistence of gastric secretion and duodenal regurgitation even after food has disappeared from the stomach.

The above relations of acidity to emptying time have already been noted by Baird, Campbell and Hern.[3]

Classification According to Gastric Tonus as found by X Ray Examination.

In Table III and Figure III thirty-nine patients have been classified by their gastric tonus as shown by independent X ray examination at the Melbourne Hospital. The results show that as gastric tonus rises, there is a simultaneous rise in the total chloride, total acidity and sodium chloride curves. Cases of achlorhydria were omitted.

TABLE III.
CLASSIFICATION OF FRACTIONAL TEST MEAL FIGURES BY GASTRIC TONUS.

Group.	Number of Cases.	Observation made.	Values at Hours.				
			½ hour.	¾ hour.	1 hour.	1¼ hours.	1½ hours.
Hypertonic ..	7	T.C.	79	88	100	103	106
		T.A.	34	46	56	58	59
		NaCl.	45	42	44	45	47
Orthotonic ..	18	T.C.	60	78	87	97	
		T.A.	32	45	46	50	
		NaCl.	28	33	41	47	
Hypotonic ..	10	T.C.	53	59	66	72	
		T.A.	27	30	37	42	
		NaCl.	26	29	29	30	
Atonic	4	T.C.	44	50	56	65	70
		T.A.	22	27	29	34	37
		NaCl.	22	23	27	31	33

Two possible explanations suggest themselves: In the first place, in the hypertonic stomach for instance, all the digestive fluids are secreted with a higher concentration of their acids, salts and alkalis. But all the available evidence is against this. That

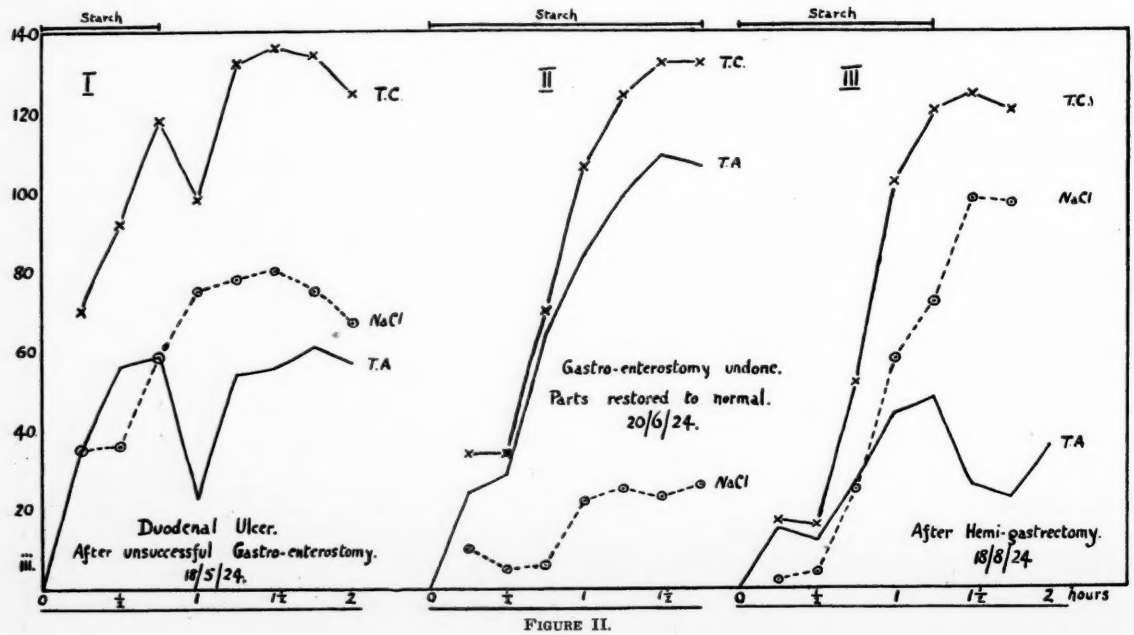

FIGURE II.

duodenal alkali and sodium chloride vary inversely and not *pari passu* is shown by the experiments and the deductions from them in the next subsection but one. The evidence against variations in secretion of gastric acid in normal people is discussed in the next subsection. In the dog, McLean[1] has shown that the concentrations of acid and neutral chloride in the gastric juice vary inversely. In the second place an inspection of the formulæ 1, 2 and 3 above and of Table I shows that there is no one factor that will simultaneously raise the total chloride, acidity and sodium chloride curves except diminution of the gastric volume $(a + b - c + d)$. It seems, therefore, that in the classes with higher tonus there is at any one moment after the commencement of the meal a smaller gastric volume undergoing chemical change than in the classes with lower tonus. This does not mean that the hypertonic class for instance will finally empty soonest. Final emptying time depends on duodenal regurgitation, as already seen. We would suggest that the early initial emptying rate of the standard 400 cubic centimetre meal, depending on gastric muscle tonus and activity and at a time when there is insufficient acid to control the pylorus, is greater than normal, leaving a smaller volume than usual into which the secretions are poured. The consequence is that a higher percentage of each value results. This explanation is supported by the observations of Campbell and Conybeare[4] that hypertonus and the rapid emptying of the barium meal, as revealed by X rays, are generally accompanied by hyperacidity with, we would add, high total chloride and sodium chloride curves as well. For further discussion on this see the next subsection but one.

The Acid Output in a Standard Test Meal.

That increased secretion of gastric acid will raise acidity, other factors remaining equal, is obvious and needs no demonstration. It is, however, doubtful if in normal men there is any appreciable variation in acid output with a standard test meal. This is supported by the following evidence:

Varying acid secretion in different stomachs would make Figure IV impossible (see under the first subsection of Section 3 below). The total chloride and acidity points would fall away from their respective curves to a far greater extent than they do. In Table VI, too, it is shown that in the vast majority of normal men the acidity value up to any moment up to one hour or more is about half that of the total chloride curve, that is to say that the amounts of acid and neutral chloride secreted by the gastric mucosa are about equal during the first hour of digestion. As the total chloride content of pure gastric juice is nearly a constant, it follows that the strength of acid secreted is nearly a constant also during the first hour.

In 1923 Apperly[5] showed that when a number of students had 250 cubic centimetres of 0·4% hydrochloric acid placed in their stomachs and the curve of acidity fall was estimated for each exactly as in

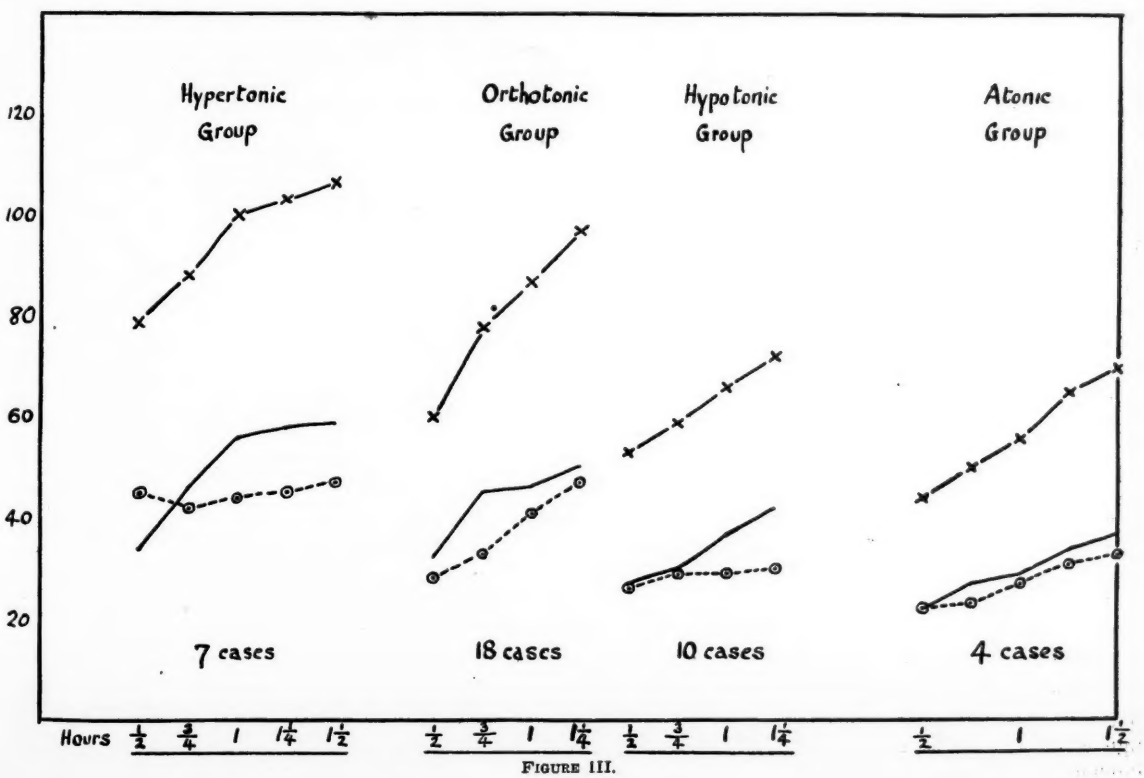

FIGURE III.

the fractional test meal and after some days these subjects were given an ordinary gruel fractional meal and the curves from both sets of experiments arranged in descending order of magnitude, that the order was the same in both sets of experiments. Variations of acid output in the gruel meal would render this impossible. The figures illustrating this are shown in the paper referred to.

Variations in the Alkali and Chloride Strengths of the Mixed Duodenal Juices.

In the duodenum secreted pancreatic alkali mixes with other duodenal fluids containing chiefly chlorides. As the former increases, therefore, the chloride strength of the mixture will diminish, that is, the duodenal alkali and chlorides vary inversely, as they do in the blood plasma from which they are drawn.

Apperly[2] has shown that when the duodenal chloride strength is high (and therefore the alkali value low) there is also in the test meal a raised value of gastric acidity and sodium chloride and therefore of total chlorides as well. The test meal findings in fact are those most commonly associated with high gastric tonus. Owing to the accompanying low duodenal alkali we would expect these stomachs to have a prolonged final emptying time, as they do.

In the same paper it was also shown that the stomach empties most readily when its contents, after neutralization in the duodenum, have a salt strength roughly equal to that of the plasma. Otherwise fluids are rejected into the stomach again from the duodenum. Consequently when the duodenal sodium chloride is high, even though the duodenal alkali be low, there will be a more rapid gastric evacuation than normally during the early part of a test meal, before there is sufficient acid to control the pylorus. There is therefore evidence in favour of the suggestion made in an earlier paragraph, that those stomachs having high gastric tonus, total chloride content, acidity and sodium chloride, have an initial rapid evacuation rate, though the total emptying time is prolonged.

This section would appear to offer some clue to the explanation of the apparently contradictory facts that, as judged by the barium meal and X rays, hypertonic stomachs empty more rapidly than low tone stomachs (Campbell and Conybeare[4]), whereas judged by the starch-iodine reaction in the fractional test meal the final emptying time of the hypertonic stomach is prolonged.

The above experiments and the deductions from them support in every way the deductions from the formulæ in Table I.

Summary.

To sum up it would appear that:

1. High gastric total chlorides, acidity and inorganic chlorides are all usually associated with high gastric muscle tonus and a high value of the duodenal chlorides and a low alkali.

2. The neutralizing mechanism (duodenal alkali strength and an efficient duodeno-jejunal neuromuscular regurgitating mechanism) determines the final gastric emptying time and not gastric tonus alone.

3. Total chloride value is determined by gastric tonus and the rate of evacuation as well as by acid secretion. Total chloride therefore cannot be safely regarded as a measure of gastric secretion as has been asserted and in fact is not so in the majority of normal cases, as has been shown.

4. It follows that the most important single factor influencing the variations is emptying time which in turn depends on the alkali output of the pancreas and on gastro-intestinal tonus. We must now proceed to show how these again depend on the chemical composition of the blood.

Section 3: Observations on the Relation of Gastric Function to the Composition of the Blood.

The Relation of Tonus, Acidity et cetera to the Bicarbonate Content of Blood Plasma.

In a recent paper we[6] described our experiments on the relation between blood chemistry and gastric test meal values. In a number of healthy fasting individuals we drew blood from a vein, "arterialized" it by saturating it with the alveolar air of one of us (K.M.S.), centrifuged it under oil and then estimated the carbon dioxide content of the plasma by Van Slyke's apparatus.[7] Immediately after drawing blood a fractional test meal was carried out. It was found, when different individuals were compared, that their total chlorides at, say, one hour varied directly as their fasting plasma carbon dioxide or bicarbonate (Figure IV). The same rule held good if these individuals were compared at half or three-quarter hours. After one and a quarter hours apparently other factors entered into the gastric mechanism which destroyed this relationship. Not only this, but the rule also held for

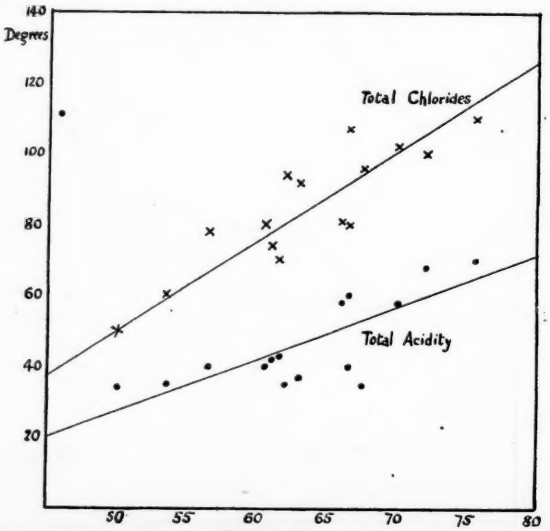

FIGURE IV.
Showing Carbon Dioxide Capacity of Plasma of "Arterialized" Blood.

acidity and inorganic chlorides. That is to say, an individual with high plasma bicarbonate has higher total chloride, acidity and sodium chloride at one hour or any other time up to one hour, than an individual with a low plasma bicarbonate. As a high value of these three substances in the gastric contents seems to indicate hypertonus, it follows that a high plasma bicarbonate commonly accompanies gastric hypertonus, while a low plasma bicarbonate commonly accompanies a low gastric tonus. This relationship was also directly demonstrated by us in the paper referred to.

In this connexion it is interesting that Bennett and Dodds[8] have also found in most of their patients a high alveolar carbon dioxide content (and therefore a high plasma bicarbonate value) in the fasting individual to accompany a high gastric acidity in a subsequent test meal.

The Relation of Gastric Acidity to the Red Cell Volume of the Blood.

For reasons to be shown later we determined during the course of our experiments the percentage volume of the red corpuscles in the blood and endeavoured to find if it was related in any way to gastric acidity. In Figure V the free hydrochloric acid at one hour after the commencement of a test meal in each of nineteen individuals was plotted against the red cell volume obtained by centrifuging the oxalated blood from each in a capillary glass tube and measuring the percentage volume of the settled cells according to the method described by Evans.[9] The figure shows in a rough way that where the cell volume is high gastric acidity and probably tonus as well are low.

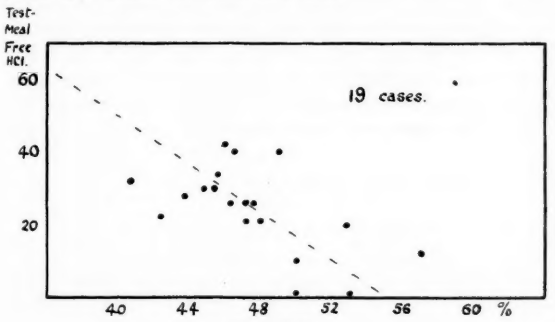

FIGURE V.
Showing Percentage Volume of Red Cells in the Blood.

The same result is shown by some experiments made by one of us (F.L.A.) some years ago. The fasting blood was drawn in a number of people in whom a test meal had been made, and the whole blood chlorides determined. The following summary shows the results. Twenty-seven people were examined and divided into three nearly equal groups. In that group in which whole blood chlorides amounted to between 0·078 and 0·086 N the highest acidity in the test meals averaged 52; the group 0·087 to 0·091 N averaged 70 and the group 0·092 to 0·097 averaged 88. For our present purposes we can regard plasma chloride as nearly constant, about 0·103 N, so that variation in whole blood chloride is due to varying cell volume. The group with a high whole blood chloride has a low red cell volume which must therefore be associated with a high gastric acidity.

Red cell volume increases in contact with a raised partial pressure of carbon dioxide. But in our experiments the latter was uniform. We therefore regard high cell volume as an indication of high cell count and increased hæmoglobin and oxygen carrying power. The significance of the inverse relation of red cell count and gastric acidity (and probably tonus) will be discussed later.

The Relation of Gastric Acidity and Total Chlorides to Acidosis and Alkalosis.

These experiments were suggested by the relationships between gastric chemistry and plasma carbon dioxide values and by the thought that the output of pancreatic alkali which so greatly influences gastric function, might depend in turn on the amount of alkali available in the blood for secretion and excretion. In these tests a series of experiments was made in which the plasma bicarbonate and cH were artificially varied and the effects of these on subsequent test meals examined. The experiments were made on three healthy individuals. The fractional test meal method was used, thin gruel being used in two men and two hundred and fifty cubic centimetres of a 0·4% solution of hydrochloric acid in the third individual. Alterations were produced in the blood by four different methods:

1. Rise of blood cH with low plasma bicarbonate by the ingestion on the previous evening of twenty grammes of ammonium chloride dissolved in 1,000 cubic centimetres of water (Haldane[10]).

2. Rise of blood cH with high plasma bicarbonate by breathing an atmosphere containing 3% to 4% of carbon dioxide for twenty to forty minutes before and during the meal (Davies, Haldane and Kennaway[11]). In some previous experiments we rebreathed our own carbon dioxide by breathing through a fifty-five centimetre (twenty-two inch) glass tube of 1·25 centimetre (half inch) bore.

3. Fall of blood cH with low plasma bicarbonate by immersion in a hot bath for twenty minutes before and during the experiment (Haggard[12]).

4. Fall of blood cH with high plasma bicarbonate by the ingestion of forty grammes of sodium acetate dissolved in one thousand cubic centimetres of water on the evening preceding the test (Haldane[13]).

Immediately before each meal the blood was drawn from a vein under oil according to Van Slyke's method,[7] and divided into two portions. One portion was equilibrated with the alveolar air of one of us (K.M.S.) and the other portion with the alveolar air of the subject of the experiment. The blood was then centrifuged without loss of carbon dioxide and the carbon dioxide and chloride content of the plasma determined by the methods of Van Slyke and Volhard respectively.

It will be noted that our estimations of plasma carbon dioxide capacity do not correspond with

those indicated in column four of Table IV. This, of course, is due to our method of "arterializing" the blood after it is drawn by equilibrating it with an alveolar air of practically the same carbon dioxide partial pressure (as by K.M.S. in column six). The more acid blood therefore takes up less carbon dioxide, while the more alkaline blood takes up more carbon dioxide. The variations in columns six and seven therefore more closely correspond with those in column three. In the hot bath experiment, for instance, our result is much higher than that which would be obtained if "arterialization" were omitted. We used this method, however, because it was the method used for making Figure IV. These reasons are given elsewhere.[6]

The clinical conditions corresponding to these experiments are indicated in column five of Table IV. The results of the experiments (Table IV) show that:

1. In the gruel meals total chloride, acidity *et cetera* do not run parallel with plasma bicarbonate, whether determined by our method or as indicated in column four of Table IV.

2. Either the duodenal neutralizing mechanism is much more effective or there is a diminished output of acid and neutral chlorides in those cases in which the cH of the blood is lowered or tends to be lowered, irrespective of the blood bicarbonate. The fact that less acid remained at one and a quarter hours after a fixed amount of acid was placed in the stomach of F.L.A. showed that the neutralizing mechanism was more effective. But this is not the whole explanation. Figure II shows that neutralization and dilution acting alone diminish the acidity without affecting the total chlorides. In the cases of G.F.S.D. and S.R., however, the alkalæmia (or the tendency thereto) following a hot bath very greatly lowers the total chloride. There must therefore be a diminished output of both acid and neutral chloride, as well as a more effective neutralization. The stomach therefore either empties more rapidly or an achlorhydria is produced. It would appear that in both the sodium acetate and hot bath experiments there is a greater excretion of alkali not only in the urine (which we found to be highly alkaline), but by the pancreas as well, with more effective neutralization of gastric acid. The above, therefore, directly support the deductions concerning variations of *D* and *bB* in Table I. The amount of free acid remaining at one and a quarter hours is given by the product of the volume remaining and its acidity at that moment (see column eleven). Clinically it is known that in the later stages at any rate, of the alkalosis following severe vomiting, examination of the vomitus reveals an achlorhydria with a high total chloride.[14] Further these experiments suggest that the fall in the acidity of the pure gastric juice towards the end of a meal, as described by McLean,[1] may depend on the alkaline tide in the blood following the earlier acid output.

3. Ammonium chloride and the breathing of carbon dioxide have effects the reverse of those under "2" above.

The height of the acidity curve seems to depend very much on volume, that is, on the rate of evacuation. Thus in the sodium acetate experiment of G.F.S.D. the acidity and total chloride curves show a sudden steep rise at the end which, however, occurs early. Presumably the extra pancreatic alkali secreted has led to more rapid emptying with a smaller gastric volume to be acidified and therefore higher acidity and chloride curves. A high salt excretion into the duodenum also leads to diminished regurgitation of duodenal fluids (Apperly[2]) which is another reason for the high acidity curve. An almost exactly opposite set of conditions occurs in the carbon dioxide breathing experiment on S.R.

In our experiments on the effect of rebreathing their own carbon dioxide by the use of a twenty-two inch glass tube, six students were tested. In the

TABLE IV.
SHOWING THE RESULTS OF ARTIFICIAL ACIDOSES AND ALKALOSES ON THREE NORMAL SUBJECTS.

1. Subject and Nature of Meal.	2. Test.	3. Blood Condition according to Various Authors. cH.	4. Plasma $NaHCO_3$.	5. Corresponding Clinical Conditions.	6. CO_2 Content of Plasma After Equilibration by K.M.S.	7. Subject of Experiment.	8. Free HCl in Test Meal at (Hours) ½	½	¾	1	1¼	9. Total Chlorides in Test Meal at (Hours) ½	½	¾	1	1¼	10. Volume Left in Stomach at 1¼ Hours.	11. Units of Free HCl at 1¼ Hours.
F.L.A. (Hydrochloric acid meal)	Normal				70	80	88	82	73	60	50	113	112	107	109	106	55	2,750
	Amm. chloride	+	−	Nephritis; diabetes	55	61	91	83	82	59	48	114	116	118	112	122	80	3,840
	Carb. dioxide	+	+	Uncompensated cardiac disease; emphysema	—	—	86	71	55	39	28	113	114	112	115	108	72	2,016
	Sod. acetate	−	+	After Sippy treatment; or severe vomiting	76	93	94	85	67	41	23	114	122	116	118	100	25	575
	Bath	−	−	Fevers; altitudes; anoxæmia; anæmia	76	78	87	73	63	25	0	120	118	116	98	105	6	0
G.F.S.D. (Gruel meal)	Normal				73	76	0	6	18	16	27	—	30	54	66	—	2	54
	Amm. chloride	+	−		49	50	11	19	39	41	42	46	52	77	74	80	10	420
	Carb. dioxide	+	+		75	—	10	17	23	28	28	49	50	59	72	80	25	700
	Sod. acetate	−	+		85	87	0	8	19	55	—	31	33	50	99	—	0	0
	Bath	−	−		—	—	0	0	0	0	0	—	—	18	18	36	55	0
S.R. (Gruel meal)	Normal				68	73	13	27	43	59	58	32	53	74	95	104	40	2,000
	Amm. chloride	+	−		55	—	5	11	15	24	32	31	46	69	70	83	120	3,840
	Carb. dioxide	+	+		63	—	0	6	15	20	25	23	23	50	53	64	250	6,250
	Sod. acetate	−	+		73	74	13	25	35	40	49	31	49	77	87	103	40	1,960
	Bath	−	−		74	75	0	0	3	11	—	17	39	37	47	—	0	0

majority the evacuation time was increased and the average acidity at one hour raised from 43 to 56, that is, by 30%. The total chloride was unaffected, probably owing to the slowed emptying time. These results are the same as in "2" above.

SECTION 4: OBSERVATIONS ON THE RELATION OF GASTRIC FUNCTION TO CERTAIN PHYSICAL AND MENTAL CHARACTERISTICS.

The Relation to Body Measurements.

Campbell and Conybeare[4] have found in general hypertonus and hyperacidity to be associated with men of the broad-chested, athletic type and the opposite gastric conditions with the long, narrow-chested unfit types. We have tried to extend Campbell and Conybeare's work, but without much success. The results obtained, however, showed a closer relationship with physical characteristics when we considered in a series of individuals the acidity and total chlorides at, say, one hour after the commencement of the meal, rather than the type of acidity chart as determined by Bennett and Ryle's method. In neither method did we find any relation between gastric function (as indicated by acidity and total chlorides) and height, weight, chest measurements, lung capacity and body area or total calories for basal maintenance as calculated from Boothby and Sandiford's charts.

Figure VI, however, shows a direct relationship with pulmonary alveolar area per unit of body weight. As the ratio of lung capacity (as determined by the spirometer) to body weight diminishes, so in general does gastric acidity at one hour or at any other time diminish when we compare a number of different individuals. The same relationship was also found to hold in the case of total chlorides and neutral chlorides at any time. (These have not been included in Figure VI because in many of the subjects total chlorides had not been estimated). From the above it appears that the larger the area of the pulmonary alveolar surface per unit of body weight, the greater the tonus and acidity of the stomach. It is possible that if the acidity value for each individual was the average of several test meals, instead of that obtained from a single test, the acidity points would have fallen within narrower limits.

The Relation to Basal Metabolism.

Table V shows a very rough parallelism between gastric acidity (and presumably gastric tonus) and the basal metabolic rate. The latter determinations were made by Dr. J. F. Chambers, using the Douglas bag and Haldane gas analysis apparatus, at the Walter and Eliza Hall Institute of Research at the Melbourne Hospital. This table would have been more satisfactory if the basal metabolic rate had been determined at about the same time as our gastric experiments were made, instead of several months apart, as they were. Again for our present purposes the average of several determinations on each individual would have been more satisfactory. Nevertheless, it is significant that the two hyper-

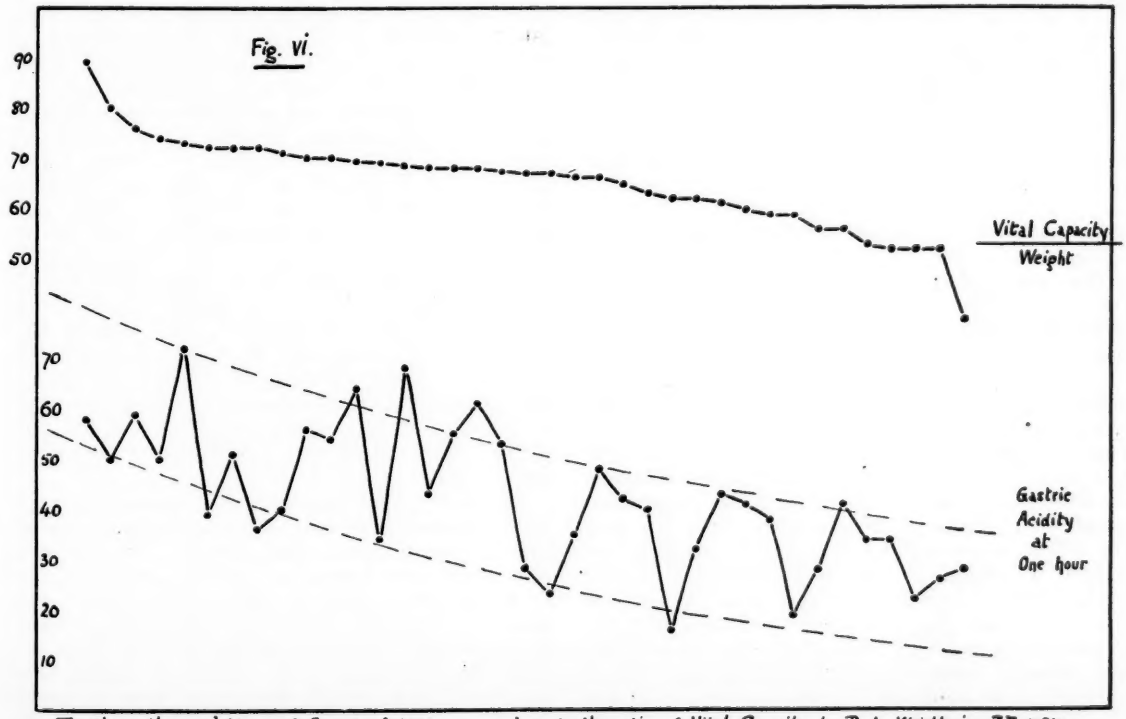

FIGURE VI.

TABLE V.
THE RELATION OF BASAL METABOLISM TO GASTRIC TEST MEAL VALUES IN TEN NORMAL STUDENTS.

Subject.	* Basal Metabolism.	Total Acidity at One Hour.	Type of Test Meal Curve (Bennett & Ryle Method).
Ed.	+ 3·3	64	Hyperchlorhydria.
Ge.	− 1·3	66	Hyperchlorhydria.
Pe.	− 2·3	24	Normal.
Na.	− 5	26	Normal.
Wi.	− 7·3	30	Normal.
Ha.	−11	54	High normal.
Gr.	−13·2	50	High normal.
Le.	−13·2	44	High normal.
Pa.	−13·4	32	Normal.
Dr.	−16·4	30	Normal.

* The basal metabolic rate determinations were made by Dr. J. F. Chambers at the Walter and Eliza Hall Institute of Research, Melbourne Hospital.

chlorhydria patients should have the highest basal metabolic rate values.

It is, of course, well known that hypertonus and hyperacidity are far more commonly seen in the athletic type, in youth rather than in age, in males rather than in females, in cold weather rather than hot weather. These conditions are those in which metabolism is raised.

Relation to Mental Ability.

One outstanding and interesting feature was the fact that the hyperchlorhydric students had by far the best scholastic records, while the "chronics" were among the low acidity figures. There were, of course, pronounced exceptions to this rule. One is tempted to speculate on the significance of this. It may indicate the sounder brain in the body which is, as we shall show later, better oxygenated than in those who, in general have a lower gastric acidity and tonus. Or again, the effect may be a vagal influence. One would expect the vagus influence to predominate over the spinal sympathetic nerves in those in whom cerebral functions are relatively stronger. A study of Figure VI favours the former view.

SECTION 5: THE RELATIONSHIP BETWEEN THESE VARIOUS FACTORS.

The association of high gastric tonus, total chloride, acidity *et cetera* with a high plasma bicarbonate may conceivably be due to one of two things.

1. The high bicarbonate or the distribution of plasma ions that accompanies high bicarbonate, may be the direct cause of a raised tonus in plain muscle. This is suggested by the experiments of Evans and Underhill,[15] of King and Church[16] and of others, showing that plain muscle tonus is increased when bathed in a plasma of increased alkalinity. It does not follow, of course, that, because the bicarbonate content of plasma is high, its alkalinity is raised. Under certain circumstances the reverse occurs, as shown in Table IV. It is almost certain, however, as will be shown, that individuals with an habitual high plasma bicarbonate do have a plasma with a tendency to be more alkaline than those with a low plasma bicarbonate. It is probable then that in these individuals the reaction of the blood does have some direct effect on the tonus of plain muscle, but we are inclined to think that this effect is small compared with that depending on oxygen supply discussed in "2" below.

2. In conditions of mild prolonged anoxæmia it is known that the tonus of plain muscle falls. This is indeed a common clinical observation. At the commencement of such an anoxæmia there is increased pulmonary ventilation with blowing off of carbon dioxide from the plasma and a fall in the $H_2CO_3 : NaHCO_3$ ratio of the plasma which therefore becomes more alkaline. To restore the ratio the kidneys excrete more alkali, hence the plasma bicarbonate falls, but a slight alkalæmia or tendency thereto remains for some time. In course of time, however, in an individual with chronic mild anoxæmia the tendency towards alkalæmia gradually changes to a definite tendency towards acidæmia, probably depending on an increased difficulty in oxidising muscle acids and on an increased venosity of the blood. In these mild chronic anoxæmia cases, therefore, we find a low muscle tonus associated with a low plasma bicarbonate value, a tendency towards acidæmia and a compensatory increase in the red cells of the blood. Conversely individuals with a high plasma bicarbonate are those with a plentiful oxygen supply and utilization, good metabolism, a high gastro-intestinal tonus *et cetera*. Gesell[17] has stated that with a normal respiratory centre high blood alkali indicates an optimum oxidation in the tissues.

To test these two possibilities, that is, that the cause of, say, low muscle tonus is due to (i) the direct influence of a low plasma bicarbonate or that distribution of ions which accompanies a low bicarbonate or (ii) a low oxygen supply leading to a faulty metabolism and a tendency towards acidæmia, the series of tests described in the third subsection of Section 3 were carried out. These had to do with the alterations in the test meal produced by artificial acidosis and alkalosis. These tests definitely dispose of the former of the two theories above advanced and support the theory that the hæmo-respiratory apparatus is responsible for the corresponding variations in gastric function and blood chemistry. Haldane[18] and others have shown that in anoxæmia there is a compensatory increase in the red cells of the blood. Our experiments show this condition to be in turn associated with low acidity which generally means poor tonus. These experiments then support the above deductions.

Alvarez[19] has shown that gastro-intestinal muscle tonus, irritability and activity depend on the rate of metabolism in that muscle. We have seen that when this tonus and activity are of a high order, gastric acidity is also usually high. Consequently we would expect some parallelism between gastric acidity and basal metabolism. We have seen that this is roughly true. Again, a higher basal metabolic rate would demand a greater oxygen supply. This could be accomplished in two ways, namely, (i) more rapid ventilation or (ii) a greater

pulmonary surface per unit mass of tissues to be oxygenated. The former of these implies a less oxygenated blood with increased stimulation of the respiratory centre. This state of affairs would be incompatible with a good metabolism. In addition increased ventilation results in blowing off of alveolar and plasma carbon dioxide and a low plasma bicarbonate. But we have already seen that high tonus and activity of the stomach are associated with the opposite set of conditions. Only the latter alternative, therefore, is possible to maintain the optimum oxygenation and our observations show that this condition, that is, a greater pulmonary surface per unit of body mass, does in fact accompany increased gastric activity. This is what we find in the broad-chested athletic type of individual. Where oxygenation is less perfect, with more rapid shallow breathing and a tendency towards acidæmia, gastro-intestinal tonus and function are less active. These associations are clinically familiar and form the basis of our classification of gastric disturbances to be discussed later.

The Authors' Theory.

Our theory connecting these observations may then be stated as follows: In those people who have long, narrow chests, with a small pulmonary alveolar area compared with their mass, oxygenation is comparatively poor. The respiratory centre and other tissues therefore become slightly more acid, producing a more rapid and more shallow ventilation. The increased ventilation again results in blowing off of carbon dioxide from the blood with diminution of plasma bicarbonate. These conditions are partly compensated by an increase in the red cell count, but full adjustment never occurs with the result that a different level of hæmo-respiratory function exists with a lower level of general metabolism. As a consequence gastro-intestinal muscle tonus, activity and irritability fall, in part as the result of the poor metabolism, possibly in part the direct result of the acidæmic tendency. In addition the loss of carbon dioxide and bicarbonate from the plasma tends to lower the osmotic pressure of the plasma. This means that plasma chlorides must be retained. Pancreatic and other glandular secretions into the duodenum therefore show a diminished chloride excretion and a preponderance of alkali in the mixed duodenal fluids. The total amount of alkali secreted, of course, must be equivalent to the gastric acid, but its relative strength in the duodenal fluids is increased. Its neutralizing power is therefore increased and owing to the combination of H and OH ions a greater fall in the osmotic pressure of the duodenal fluids results. This leads, as Apperly[2] has shown, to increased regurgitation into the stomach, dilution of acid there and more rapid evacuation of the stomach, in spite of its poor tonus and activity.

On the other hand, with good oxygenation and high gastric tonus there seems to be an early rapid evacuation of gastric contents before any appreciable acid has been secreted. This effect seems to be purely of muscular origin before there is sufficient acid secreted to control the pylorus. Hence the various secretions, mixing with a smaller gastric volume, produce a higher percentage of total chloride acidity *et cetera* than normally. Later, however, as acidity rises, acid chyme entering the duodenum meets duodenal fluids of low alkali value, so that only slow neutralization occurs. Apperly[2] has previously shown that regurgitation from the duodenum into the stomach in these circumstances is diminished. Acid lies, therefore, longer unneutralized in the upper part of the duodenum and keeps the pylorus closed for longer periods with delayed final emptying, in spite of the early rapid emptying rate. The diminished regurgitation from the duodenum still further leads to high gastric acidity.

From the above then it would seem that gastric function depends on blood and oxygen supply and on the pH of the plasma. It will therefore be easy to understand how pathological conditions, associated with variations in these conditions, will lead to disordered gastric function which can be revealed by the fractional test meal. The fractional test meal, therefore, can be used to give information concerning changes in the blood, their nature and a general indication of the cause or causes of these.

Section 6: Clinical Examples.

In a recent paper Apperly[20] proposed a new classification of the non-organic dyspepsias based on some of the principles here set out. He pointed out that that group of symptoms which we may call the "regurgitative group" (eructations, belching, heartburn, nausea, anorexia, fullness soon after meals, biliousness, vomiting *et cetera* according to Alvarez[19]) which is known to depend upon a low gastric tonus and to be associated with hypoacidity, is produced by any condition interfering with blood and oxygen supply to the gastric muscle. These conditions are:

1. General.

(*a*) Faults in oxygen supply — balloon and mountain sickness, carbon monoxide poisoning, side-tracking of oxygen supply from the digestive tract to voluntary muscles as during severe exertion (the "physiological dyspepsias").

(*b*) Faults of oxygen carriage—chronic heart and lung conditions, anæmias, deficient exercise with habitual shallow respiration.

(*c*) Faults in the tissues leading to ineffective utilization of oxygen—fevers, toxic states, chronic nephritis, diabetes, pulmonary tuberculosis, fatigue, general debility, hypothyreoidism.

2. Local.

Diminished lumen of gastric vessels (overstretching following habitual overeating and gastroptosis, arterio-sclerosis); hepatic cirrhosis, with gastro-intestinal congestion.

On the other hand, gastric tonus and activity are increased by increased general metabolism, as in youth, cold weather, males rather than females, athletic types and following healthy exercise. The clinical evidence, therefore, strongly supports the authors' theory.

SECTION 7: WHAT IS A NORMAL TEST MEAL CHART?

We have seen that no special values of total chlorides, acidity *et cetera* can be regarded as normal. The range of each of these is as wide in health as in disease. What then can we regard as a normal test meal chart?

We have seen that the form of the chart is the result of two sets of factors: (i) the secretion of acid, salts and alkali, depending in non-organic conditions on the amounts of these available in the blood for secretion and excretion, (ii) the neuro-muscular mechanism of the gastro-intestinal tract, producing a flux and reflux of gastro-duodenal contents. The normal relationship of these is such that during the rise of the acidity curve, usually extending over the first one to one and a quarter hours, the sodium chloride curve corresponds fairly closely to the total acidity curve, that is, each of these is at any moment up to one or one and a quarter hours half the value of the total chloride curve, no matter what the actual values of each. This is shown well in Figure IV. After one to one and a quarter hours the acidity curve falls slowly to zero, while the sodium chloride curve rises steadily till it reaches the total chloride curve.

Now dyspeptic symptoms arise when the normal coordination of the neuro-muscular apparatus producing flux and reflux is altered. One part of the muscular apparatus is opposed to the action of another part, for example, the attempt of the stomach to empty with a pyloric spasm and alterations of pressure or tension on the contents are produced. This gives rise to the sensations of fullness, discomfort and pain. We would therefore expect this incoordination to be reflected in the ratio between the total chloride and acidity (or sodium chloride) curves. In Table VI are shown the proportions of normal students and dyspeptics in whom the total acidity value up to one hour is about half the total chlorides or otherwise. In the dyspeptic group are included all patients sent for test meal at the Melbourne Hospital over a certain period in whom total chloride estimations were made. We have taken all patients in spite of the fact that many suffering from asthma, rheumatoid arthritis and others were included, because it has not been possible to delete all the non-dyspeptics. It is therefore certain that the figure 54% is too high.

TABLE VI.

NORMAL AND DYSPEPTIC TEST MEAL CHARTS CLASSIFIED ACCORDING TO THE PROPORTION BETWEEN SODIUM CHLORIDE (OR TOTAL ACIDITY) VALUES UP TO ONE HOUR (OR HIGHEST POINT OF ACIDITY) AND THE TOTAL CHLORIDE VALUE AT THE SAME TIME.

(All figures given as percentages.)

Subjects.	T.A. and NaCl Curves fall in Area bounded by ½ and ⅔ T.C. Values.	NaCl Curve falling below ½ of T.C. Values.	NaCl Curve falling above ⅔ of T.C. Values.
Normal students (52 men)	87	6	6
Dyspeptics and some others (82 men and women)	54	34	12

A glance at Figure III is sufficient to show that the cause of this incoordination is not alterations in tonus, since no matter what tonus may be, the 2 : 1 ratio still holds good between total chloride and acidity. But Figure I seems to show that the fault lies somewhere in the apparatus responsible for neutralization and dilution such for instance as pyloric spasm or an altered activity of the regurgitative mechanism. Of these, Table VI shows that causes producing failure of regurgitation are the most numerous. We hope to deal with this aspect of dyspepsia, however, in a later communication. It would seem then that a normal test meal chart must show three things: (i) a fall in the acidity curve following the preliminary rise, (ii) a 2 : 1 ratio between the total chloride and acidity curves during the rise of the latter, (iii) an acidity value at one hour within the limits corresponding to the ratio of vital capacity to body weight, as indicated in Figure VI.

CONCLUSIONS.

1. It is impossible to interpret the fractional test meal curves unless: (i) total chloride, acidity and inorganic chloride curves are recorded, (ii) we have some knowledge of the effects of variations of secretion of acid, alkali and salts and of gastro-intestinal muscle on the curves mentioned.

2. Observations and experiments indicate that: (i) provided the gastric musculature is efficient, emptying time is determined solely by the alkalinizing mechanism of the duodenum. (ii) Total chloride values, acidity and inorganic chlorides vary with and are a fair indication of gastric tonus and activity which in turn varies with the bicarbonate strength of the blood plasma, general metabolism and the ratio of total lung capacity to body weight and inversely as the red cell volume of the blood. (iii) As a rule high values of the factors mentioned under (ii) are accompanied by a slowly emptying stomach (final time). (iv) Provided the total chloride and acidity values bear roughly a 2 : 1 ratio, gastric acidity is determined chiefly by gastric muscle tonus and activity. With other ratios variations in acidity appear to depend on an altered pH of the blood or a tendency thereto.

3. The authors' theory connecting these facts is as follows: That general bodily health, as reflected in gastric and other muscle tonus, depends on good metabolism and oxygen supply. When for any reason there is a condition of anoxæmia in the tissues, poor gastric tonus results. There is also an increased pulmonary ventilation with a rise in the red cell volume of the blood, a fall in the alveolar carbon dioxide and plasma bicarbonate, an increased alkali value in the duodenal fluids with more rapid emptying, greater regurgitation into the stomach and lower acidity, total chlorides and inorganic chlorides. Any alteration in the normal relationship between the various constituents of the blood is reflected in the behaviour of the gastro-intestinal musculature and secretion. Many of the dyspepsias depend on these altered functions.

4. Based on the above and on the gradient theory of Alvarez a new classification of the non-organic dyspepsias is suggested.

5. The question of what constitutes a normal gastric function, as shown by the fractional test meal chart, is discussed.

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THE FRACTIONAL TEST-MEAL IN NORMAL STUDENTS: A COMPARISON OF RESULTS WITH THOSE OF OTHER OBSERVERS.

By FRANK L. APPERLY, M.A., M.D. (Oxon.), D.Sc. (Melb.),
Honorary Physician to Out-Patients, Saint Vincent's Hospital, Melbourne; Senior Lecturer in Pathology, University of Melbourne;

AND

KATHLEEN M. SEMMENS, B.Sc. (Melbourne).
[From the Macleod Laboratory, Pathology Department, University of Melbourne.]

PART of the data in our paper on variations in normal gastric functions and their causes was obtained from observations of normal students. These results, together with those of certain earlier experiments, are of interest from the point of view of comparison with those of other workers. In this paper are recorded our own averages, comparisons of these with those of other workers and the results obtained by combining our results with those of others.

Our figures for Australian students are unfortunately not so complete in some respects as those of Bennett and Ryle[1] and of Baird, Campbell and Hern[2] [5] for English students. On the other hand, we are able to include a larger series of total chloride curves than that of the last-named workers who are the only ones who have hitherto published results of this nature.

Results of Classification into High, "Normal" and Low Acidity.

In the classification of results into high, "normal" and low acidity we have followed the method of Bennett and Ryle[1] entirely, using their diagram for classifying our own men. In all we examined ninety young men, all between the ages of nineteen and thirty. These were all healthy medical students, except a few who were healthy young patients after discharge from the Melbourne Hospital suffering from healed fractures and wounds. The results are shown in Table I. Of these ninety men, seven

TABLE I.
CLASSIFICATION OF A NUMBER OF NORMAL YOUNG MEN BY THEIR SECRETORY CURVES.
(Figures given as percentages.)

Observers.	Number of Cases.	Achlorhydria.	Hypochlorhydria.	Low Normal.	Normal.	High Normal.	Hyperchlorhydria.
Bennett & Ryle [1] ..	100	4	1	10	59	18	8
Baird, Campbell & Hern [2]	57	2	7	26	40	14	11
Apperly & Semmens ..	90	8	4·5	5·5	42	19	21
Above groups combined	247	5	4	12	49	17	13

had achlorhydria and of these we shall speak later. Figure I has been constructed from the test meal charts of the remaining eighty-three men, according to Bennett and Ryle's plan, only the free acidity being plotted. Of these curves 50% fall within the area between the lines C and D, while B and E enclose an area holding 80% of all curves. With one exception (a curve rising to F and G) the free acid curves of all cases fall between the line A and zero.

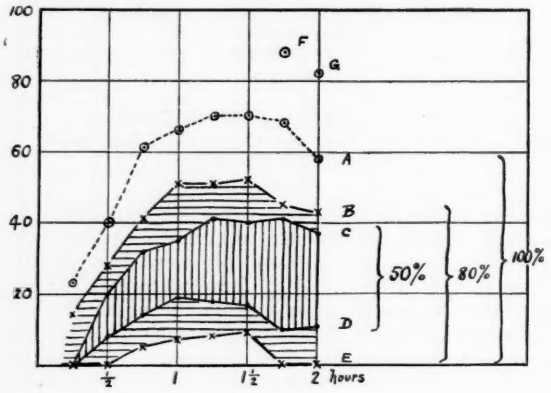

FIGURE I.

On comparison of these results with those of Bennett and Ryle it will be seen that both our 50% and 80% areas are slightly broader and at a higher level than theirs. The limits are about the same, but our average acidity is rather above theirs. These differences appear to be due to our higher proportion of persons with hyperchlorhydria (Table I) and this again may possibly be explained as follows.

It is a common observation that hyperchlorhydria is much more common among the higher intellectual types than among others of less intelligence. Our first twenty students were wholly "honours" men and scholarship men, the remainder being nearly all "pass" men. It was the former group that supplied the great majority of those with hyperchlorhydria, thirteen of them being high normals or persons with hyperchlorhydria. Among the public hospital class hyperchlorhydria is comparatively rare in our experience. It is for this reason that we are inclined to criticise Bell's[4] comparison of the proportions of hyperchlorhydria, hypochlorhydria *et cetera* among known diseases, with the proportions of these among normal students. Bell's patients were drawn from both public hospital and private patients.

Again in our group of ninety men there were seven with achlorhydria. Against the criticism of Campbell[5] that achlorhydria found among normal students might not be true achlorhydria are the facts that: (i) student J was twice found to have achlorhydria; (ii) student M also had achlorhydria on each of two examinations and his father died of pernicious anæmia (it is well known that achlorhydria and other gastric types tend to run in families); (iii) student R at one examination had achlorhydria and his mother, suffering from rheumatoid arthritis, gave us a similar result a year before. We therefore regard these men as having congenital achlorhydria. The total chloride in J was 40° and in R 78° at one hour, the latter an unusually high figure if a true achylia. The remaining four students were examined once only. We have noted in our experience of normal and hospital patients a curiously high proportion of the dark-brown-eyed type among achlorhydrias. What is the significance of this?

Total Chlorides.

Our results for total chlorides have been plotted in very much the same way as were the acidity figures just given. In Figure II the area between curves C and E includes 50% of all total chloride curves, while the 80% and 100% areas are included between curves BF and AG respectively. Curve D is the average of all. The numbers of individuals used in estimating these values are indicated at each point of time along the uppermost curve A. All those with achlorhydria were omitted. As many of these tests were made for other purposes (see our previous paper) some were ended at one and a half hours. Hence the curves after this point are made up from smaller numbers. The fact that the average total chloride curve finally reaches a value nearly equal to the chloride strength of plasma (0·103N) has been noted and its probable significance commented upon elsewhere (Apperly[3]).

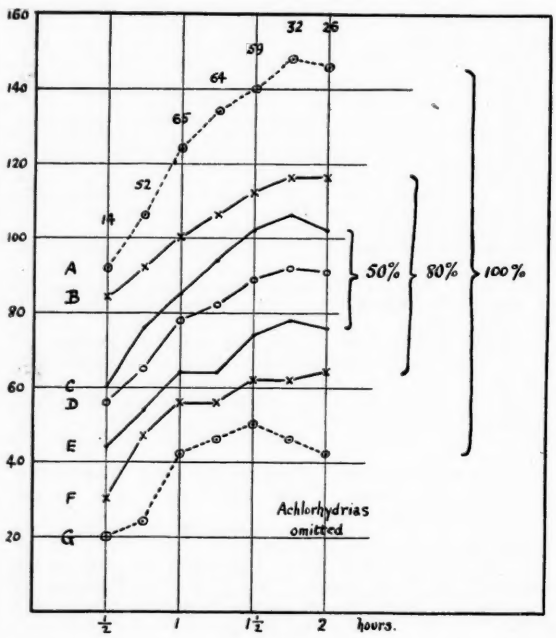

FIGURE II.

Baird, Campbell and Hern[5] are the only other workers who have, so far as we are aware, published similar results for total chlorides. In their paper they give the results for thirty students. Our curves, however, are much higher than theirs and

the 80% area is broader. We estimated total chlorides by Volhard's method, using clear filtered or centrifuged fluid, as they did. Figure III shows the area constructed by combining our 80% area with their 80% area. It does not follow of course that 80% of our combined cases fall within this area, but the figure is probably a fairly close approximation.

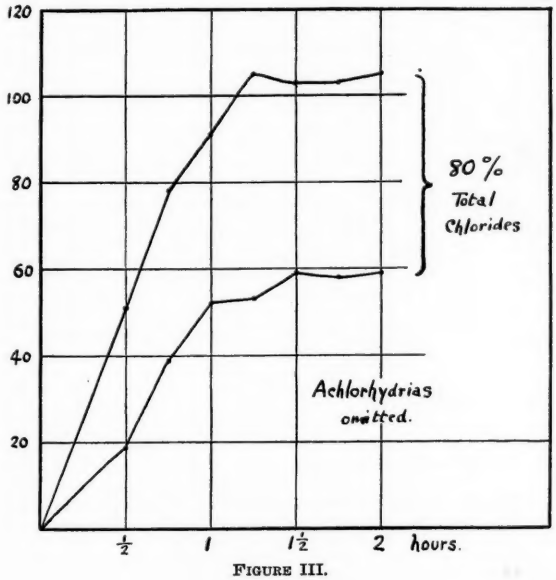

FIGURE III.

Emptying Time.

Table II shows our subjects classified according to their emptying time as judged by the disappearance of the starch-iodine reaction. For comparison the results of other workers are shown and the proportions found in a combined total. None of our estimations was taken beyond two hours, so that our results are not so complete as the others. The most extraordinary point is that in spite of our high proportion of hyperchlorhydria, we should have so small a proportion of men whose stomachs emptied at more than two hours. Owing to the fact that the starch-iodine test was omitted in a number of our subjects, the number of men in our series in Table II is smaller than that in Table I.

TABLE II.

RATE OF EMPTYING OF THE GRUEL MEAL.

(Figures given as percentages.)

Observers.	Average Emptying Time.	Stomach contains no Starch at (Hours)					
		0–1	1¼	1½	1¾	2	2+
	Hours.						
Bennett & Ryle [1] (100 men) ..	1·9	6	3	7	13	17	54
Baird, Campbell & Hern [2] (57 men)	2·1	5	7	14	9	17	48
Apperly & Semmens (53 men)	—	2	9	11	25	17	36
Above groups combined (210 men)..	—	5	6	10	15	17	47

References.

(1) T. I. Bennett and J. A. Ryle: "Studies in Gastric Secretion: A Study of Normal Gastric Function of One Hundred Healthy Men by Means of the Fractional Method of Gastric Analysis," *Guy's Hospital Reports*, 1921, Volume LXXI, page 286.

(2) M. McC. Baird, J. M. H. Campbell and J. R. B. Hern: "Gastric Secretion, Physique and Physical Fitness," *Guy's Hospital Reports*, 1924, Volume LXXIV, page 339.

(3) F. L. Apperly: "Duodenal Regurgitation and the Control of the Pylorus," *The British Journal of Experimental Pathology*, June, 1926, page 111; "The Use of Hydrochloric Acid and Sodium Bicarbonate in the Treatment of Certain Common Dyspepsias," THE MEDICAL JOURNAL OF AUSTRALIA, March 27, 1926, page 354.

(4) J. R. Bell: "Notes on a Consecutive Series of 425 Gastric Analyses by the Fractional Method," *Guy's Hospital Reports*, 1922, Volume LXXII, page 302.

(5) M. McC. Baird, J. M. H. Campbell, and J. R. B. Hern: "The Importance of Estimating Chlorides in the Fractional Test-Meal Samples, and Some Experiments with the Duodenal Tube," *Guy's Hospital Reports*, 1924, Volume LXXIV, page 23.

Reports of Cases.

IDIOPATHIC NARCOLEPSY.

By JOHN BOSTOCK, M.B., B.S. (London), D.P.M.,

Honorary Neurologist, the General Hospital and Mater Misericordiæ Hospital, Brisbane.

SINCE Dr. W. J. Adie's article on idiopathic narcolepsy, references to this interesting disease are becoming more frequent. The condition is, however, sufficiently rare to warrant publication of further cases with an additional note as to the progress of the patient, T.C., described by me in THE MEDICAL JOURNAL OF AUSTRALIA on August 20, 1927.

For those who have not recourse to the original description it may be mentioned that idiopathic narcolepsy is characterized by periodic attacks of intense sleepiness and cataplexy on emotion.

By the couretsy of Dr. E. B. Fitzpatrick, of Tamworth, I am permitted to record the following case.

G.K., *ætatis* forty-three, a married woman, consulted him on September 20, 1927, concerning certain "turns" she experienced. The first was three years previously on her hearing that her sister's premature twins had died. A year later she had a second attack after being frightened by one of her children. In July, 1927, she felt herself becoming very drowsy and would go to sleep in the daytime. A month later if she laughed her head would nod and her arms and legs collapse. She would fall to the ground. The "turn" lasted one to two minutes. Excitement did not affect her, since she was able to go to the races and see her horse win without untoward symptoms, but on laughing about it afterwards she would have a cataplectic seizure. She was conscious of its onset and had time to ask friends to support her.

When she consulted Dr. Fitzpatrick the above attacks averaged four or five *per diem* and the sleepiness was increasing. Driving in a car she would drop off to sleep. Her sleep did not refresh her and in the morning it was an effort to get up. The "turns" were worse at the menstrual periods and on September 25 fifteen "turns" occurred between 2 p.m. and 4 p.m.

Suggestive treatment was tried without effect and on October 13 following the treatment suggested in the case of T.C., a mixture containing caffeine citrate 0·3 gramme (five grains), *liquor strychninæ* 0·6 mil (ten minims), tincture of opium 0·54 mil (nine minims) was exhibited thrice daily. The patient expressed herself as highly delighted with the result, since she lost the sleepy feeling and her cataplectic attacks became very infrequent and indeed so mild that only previous experience enabled her to recognize them.

Dr. Fitzpatrick made the additional interesting observations that the patient dreamed a great deal after treatment

and often woke up with a start. Diminution in the dose of medicine caused her to become more sleepy.

B.B., *ætatis* thirty-two years, was referred to me by Dr. L. J. J. Nye on account of certain curious symptoms. His "nerves" would let him down in playing a difficult shot at tennis. When fishing he could not pull a fish out of the water because his muscles became limp. If he saw a cat, he could not chase it. If he laughed, all his muscles seemed to drop; this commenced in the mouth and he had often fallen to the ground. The attacks lasted for about thirty seconds. On inquiry I found that sleep attacks were also present. He has often had to stop his motor car and get out on any excuse, for example, to buy cigarettes. The walk banished the sleep tendency. If he sat in a chair and talked for a while, he fell asleep. The sleepy feelings came on at any time, but were most frequent between the hours of ten and eleven in the morning and after meals.

The patient first noted cataplectic attacks just after the war; he could then control them by holding his jaw. During war service he had been blown up by a shell, but had not suffered from concussion. There had been no head injury.

On examination he was found to be of the intelligent, matter-of-fact type and showed no tendency to hysteria. Physically he was well built, all organs were apparently healthy. The cranial nerves were normal. The wrist jerks were brisk, but the knee and ankle jerks were sluggish. A skiagram of the skull revealed normal contours.

For treatment a mixture containing caffeine citrate 0·18 gramme (three grains), tincture of opium 0·36 mil (six minims), tincture of *nux vomica* 0·48 mil (eight minims) was given and the patient expressed astonishment that he was able afterwards to play a hard game of tennis without having any seizures and his sleepiness was lessened. Unfortunately he left the country a few weeks later, so that no subsequent results of treatment can therefore be given.

This case illustrates the need for further prognostic criteria. The patient asked if marriage was advisable. I could only tell him as the number of recorded cases was so few, it was possible but by no means certain that his complaint was compatible with a reasonably long working life.

The next case is less typical, but I have included it in the series because of definite attacks of sleepiness together with phases in which the patient would collapse and fall to the ground without loss of consciousness. The exciting cause of the latter was abdominal pain for which incidentally no cause was found. This patient illustrates the fact that positive symptoms such as grimacing, protrusion of the tongue and stammering, mentioned by Adie, may be present. M.H. complained of twitching of his muscles, particularly in the right side of the face. This type of narcolepsy may be mistaken for epilepsy and it is probable that quite a number of epileptics should have been labelled narcoleptics.

M.H., *ætatis* thirteen, was seen by me at the Mater Misericordiæ neurological clinic on December 28, 1927. He complained that for three years he had had about once a month attacks of severe pain on the left side below his ribs during which he would collapse to the ground and had to be taken to bed. They were accompanied by twitching of his muscles, particularly in the right side of his face. In addition he was frequently sleepy and would fall asleep during the day. On one occasion after hearing thunder, he lost the power of speech for ten minutes and afterwards vomited. There was an admission of having always been frightened by storms. Occasionally on laughing heartily it was necessary for him to sink into a chair. On account of the above symptoms, his attendance at school had been very irregular.

His previous health, babyhood, had been uneventful, but at the age of five he had had pneumonia and had fallen down some steps, seriously injuring himself. The attacks had commenced about this time.

He was the eldest of five, one brother died of meningitis and a sister of pneumonia; the others are healthy. An uncle is said to have had general paralysis of the insane. Mentally the boy was "bright" and his outlook appeared to be normal. He had attained the fifth class at school. Physically he was tall and spare. His blood pressure registered 110 millimetres of mercury. Pulse rate was 96. There were no adventitious sounds. The chest and abdomen appeared normal. The cranial nerves showed no abnormality. The jaw jerk, wrist jerk and abdominal reflexes were definitely increased; knee and ankle jerks were active. Plantar reflex was flexor. On the sensory side root areas of anæsthesia to prick were present, involving eleventh dorsal to third lumbar roots on the left side and twelfth dorsal to second lumbar on the right side. The blood was subjected to the Wassermann test, but yielded no reaction.

After treatment by caffeine citrate, tincture of *nux vomica* and tincture of opium he showed a definite improvement, is more lively and has had neither catalepsy nor facial spasm.

Dr. H. M. North in a letter, dated November 15, 1927, kindly gave me further particulars of the subsequent history of T.C. He was still receiving treatment by caffeine, opium, strychnine and admitted to be much improved, though he still experienced several sleepy turns each day. He noted that he now takes size eight and a half in boots instead of seven and a half, and Dr. North was of the opinion there was a more or less generalized skeletal overgrowth pointing to pituitary disease. An interesting symptom was noted, namely, that the patient often dreams of a smell like that of corpses or that he is vomiting very copiously, though he cannot remember ever having actually vomited in waking life. The dreams are reminiscent of the olfactory auræ in the dreamy state of hippocampal fits, although not, of course, identical.

Comment.

Adie remarks that:

> Many forms of treatment have been tried without success: bromides and other anti-epileptic remedies produce no favourable effect. Some have tried to diminish sleep during the day by making the night sleep more profound, others have given caffeine and other drugs reputed to prevent sleep. Thyroid and various polyglandular preparations have also been used. In many instances the attacks became less frequent while the patients were resting in hospital, but they recurred with the original frequency soon after discharge.

In the present series of cases the exhibition of caffeine, strychnine and opium appears to have had a definite beneficial result without recourse to in-patient treatment, in checking to some extent the sleepiness and to a greater degree the cataplexy. It is perhaps unfortunate that opium was used in combination with the other drugs, since it is probably the most useful. If the conception of narcolepsy as a peculiar kind of nervous activity that allows excessive responses to emotional stimuli and betrays an undue fatigability of nerve cells be accepted, then the morphiates would be strongly indicated. In psychiatric practice which deals very largely with functional emotional excesses, their utility is almost universally exploited in certain conditions. Moreover, they help to avoid neural fatigue. This latter factor is probably of more importance than is bestowed on it in Dr. Adie's monograph, since he lays the greater stress on inhibition, though the difference may be more apparent than real, it is difficult to divorce inhibition from fatigue.

Dr. Lind's letter to the journal of September 3, 1927, deals with a good example of what might be termed physiological narcolepsy, fatigue of the nervous system due to deficient oxygenation and excessive emotion owing to difficulties in respiration. It would be interesting to know if morphine has ever been employed by mountaineers or aviators in their attempts on altitude records.

Summary.

1. An account is given of three cases of idiopathic narcolepsy with progress notes concerning a fourth previously reported.

2. Further notes are made as to the utility of caffeine, strychnine and opium in treatment.

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1. W. J. Adie: "Idiopathic Narcolepsy: A Disease *Sui Generis;* with Remarks on the Mechanism of Sleep," *Brain*, Volume XLIX, Part 3, 1926, page 257-306.

2. John Bostock: "Idiopathic Narcolepsy," THE MEDICAL JOURNAL OF AUSTRALIA, August 20, 1927, page 258.
3. W. A. T. Lind: "Idiopathic Narcolepsy," THE MEDICAL JOURNAL OF AUSTRALIA, September 3, 1927, page 351.

AN UNUSUAL CASE.

By RUPERT A. WILLIS,
Medical Superintendent, Austin Hospital, Melbourne.

Clinical History.

A.M., MALE, single, aged twenty-six years, was admitted to the Austin Hospital on December 9, 1926, with a history of weakness, loss of weight and cough for twelve months and some firm swellings on both sides of the neck for three months.

On examination he was an anæmic, but fairly well nourished man. There were gross signs of fibrosis and cavitation of the upper lobe of the left lung, fibrotic signs in the right lung and râles and crepitations at both apices. The heart was apparently normal, save for a definitely accentuated second sound at the pulmonary area. Abdominal examination revealed no abnormality; the liver and spleen were not evidently enlarged. There was no neurological abnormality. On each side of the neck in both anterior and posterior triangles there were a number of firm, painless, discrete, enlarged lymph glands, unattached to adjacent structures, the largest being fully three centimetres in diameter. There was slight enlargement of inguinal glands, but no axillary glands were palpable.

Special examinations were carried out.

The sputum contained many tubercle bacilli. The blood manifested a moderate grade of secondary anæmia, non-distinctive in character.

Radiography of the chest revealed extensive fibrosis of both lungs and a left apical cavity. An oblique picture also disclosed several large discrete shadows of posterior mediastinal glands.

On February 3, 1927, a full single application of deep X rays was made to the neck and definite reduction in the size of the enlarged glands occurred. No subsequent further enlargement of the cervical glands developed. During the six months following his admission the patient remained in fairly good general condition, eating and feeling well and maintaining his weight. He had, nevertheless, during the whole time a prominently swinging temperature, averaging between 36·1° and 36·6° C. (97° and 98° F.) at 6 a.m. and about 38·8° C. (102° F.) at 6 p.m. This was unaccompanied by either shivers or sweats and was in striking contrast with the patient's relatively good general health. His condition was regarded latterly as Hodgkin's disease in addition to pulmonary tuberculosis.

On August 22, 1927, he developed headache, drowsiness and photophobia and in a few days neck rigidity occurred and Kernig's sign was elicited. The cerebro-spinal fluid was under increased pressure, was slightly clouded, contained a fair number of lymphocytes, increased globulin, but no bacteria were found. He died on September 1, 1927.

Post Mortem Findings.

At *post mortem* examination both lungs were everywhere densely adherent to the parietes and were the seat of universal fibrosis, with scattered tubercles and fibrous-walled cavities at both apices. The cervical glands were discrete, pale, firm and on section presented a firm yellowish tissue with no softening. Several enlarged posterior mediastinal glands were exactly similar and there was considerable enlargement of gastric, pyloric, hepatic, prepancreatic and mesenteric glands, all similar in character to the cervical glands. The glands in the lesser omentum and hilus of the liver were specially enlarged, surrounding and obscuring, but not attached to the common bile duct and portal vein. Pelvic, inguinal and axillary glands appeared normal. The brain was affected by basal and lateral lepto-meningitis, with much firm exudate and conspicuous tubercles of the *pia mater* of the temporal lobes and cerebellum. The foramen of Magendie was occluded and the ventricles dilated.

Microscopical examination of sections revealed normal liver, spleen, pancreas and kidneys, fibro-caseous tuberculosis of all enlarged lymph glands with many giant cells, typical chronic tuberculous changes in the lungs and tuberculous infiltration with many giant cells of the meninges. Tubercle bacilli were demonstrated in sections of the lungs, glands and brain.

Comment.

Features of interest in the case are:

1. The widespread chronic tuberculous adenitis, which clinically and even *post mortem* strongly suggested Hodgkin's disease.
2. The response to X ray therapy.
3. The terminal meningitis, unusual in an adult and unassociated with any evidence of generalized miliary dissemination.

Bibliography.

Osler's "Principles and Practice of Medicine," Tenth Edition, page 177.
Mallory: "Principles of Pathologic Histology," 1923, page 333.

Reviews.

TUMOURS.

A THIRD edition of Ewing's well known book on neoplastic diseases, now to hand, has been awaited with interest, because six years have elapsed since the issue of the second edition and much valuable work on new growths has been published during this period.[1] About sixty new illustrations—gross, microscopical and radiographic—have been added. The author considers that photographs are better for illustration than drawings. With this we entirely agree. For teaching purposes drawings are often better, because they show outlines and structure more clearly, but when the reader wishes to form his own opinion as to the microscopical nature of a tumour, the next best thing to studying the original section is the examination of an untouched photomicrograph. The book is in the nature of a compilation of views of various authorities with occasional paragraphs in which the author expresses his own opinions. When reading the book one feels at times that it would be improved if the author's views were given greater prominence and those of other authorities were curtailed.

When dealing with the parasitic theory of the origin of cancer the author mentions the work of Gye and Barnard which recently caused such a stir, and like G. W. Nicholson and many other authorities, is sceptical concerning it; Ewing considers that Gye's theory stands at present without substantial basis. Under the heading "Chemistry of Tumours" Warburg's studies on the glycolytic properties of tumour cells are referred to. The metabolism of resting epithelium under aerobic conditions with normal respiration is an oxidation metabolism, not a splitting metabolism. The respiration of normal tissues suffices to bring about the disappearance of the glycolytic products, whereas in tumours the respiration is too small for this purpose and these products accumulate and injure the organism. Warburg conceives, perhaps without adequate grounds, that in every tissue there are some cells which glycolize actively while the others act feebly and that as the result of oxygen deficiency from injury or irritation, these active cells divert nutriment and grow at the expense of others. Oxygen deficiency continuing, there will arise a tissue with high glycolytic power and low respiration, that is, tumour tissue. Warburg finds that radiation reduces the glycolytic power of tumour cells and he attributes some of the favourable effects of radiation therapy to this factor.

The chapter on tumours of bone has been entirely rewritten on the basis of information obtained largely from the Codman Registry of Bone Sarcoma and several

[1] "Neoplastic Diseases: A Treatise on Tumors," by James Ewing, A.M., M.D., Sc.D.; Third Edition, Revised and Enlarged; 1928. Philadelphia: W. B. Saunders Company; Melbourne: James Little. Royal 8vo., pp. 1127, with illustrations. Price: 63s. net.

new subvarieties of these tumours have been formally recognized. The subject of brain tumours has been recast, brought into line with recent contributions in this field and the newer nomenclature used by Cushing and Bailey and others has been partly adopted.

Ewing is non-committal when discussing melanomata, but he mentions Dawson's recent elaborate monograph in which nævus cells were traced to the basal epithelium of the epidermis thus confirming Unna's views.

The author's views as to the nature and origin of Paget's disease of the nipple are unaltered. That Ewing considers the changes in the epidermis, in at least some cases, to be due to changes in epithelial cells *in loco* is clear from his illustrations, two of which are taken from Fordyce's case of so-called Paget's disease of the buttock. In the second edition of this book Ewing stated that commonly included under the term Paget's disease are cases of diffuse carcinoma of the breast which invade the skin at the nipple and extend widely over one or both breasts often with marked hyperæmia. In the latest edition (third) this statement is omitted and the photograph which illustrated the condition and which appears remarkably like cancer *en cuirasse* of Velpeau has been transferred to another part of the book (Figure 256, page 574). Figure 427, page 872, which is said to be a duct carcinoma associated with paget's disease, is ill chosen. There is nothing characteristic about it and the duct which is so conspicuous does not show the stuffing of the lumen with cancer cells which is so striking in Paget's disease.

A criticism which applies to at least many of the illustrations in the book is that there is no reference to them in the text. Where rare conditions are illustrated, this omission is a decided drawback. Presumably it is assumed that the illustrations are characteristic of definite types of new growths, but in many instances this is far from being the case.

Though Bowen's precancerous dermatosis is mentioned by Ewing in this edition for the first time, the reference to Bowen's original article could not be found in the bibliography.

Because our knowledge on many fundamental points of importance is so meagre, a book of this nature is difficult to write and in spite of the few minor criticisms that have been offered, we think that the author has achieved his chief object that, by rendering more accessible to English readers the knowledge of tumours, he may contribute something towards the reduction of the mortality of cancer.

TUBERCULOSIS IN DISGUISE.

EVER and anon the profession of medicine is more or less disturbed by one of its number who, perhaps in all sincerity, perhaps solely as a "stunt," offers to a credulous world a new and simple cure for some disease, the accepted treatment of which has hitherto been tedious and fraught with uncertainty both to patient and physician. Tuberculosis is one of the happy hunting grounds of these enthusiasts.

Into this field there comes one, Joseph Hollòs, M.D., of New York and late of Budapest. He presents to us "Tuberculous Intoxications, a Clinical Study."[1] With a wide flourish he takes for his purpose a number of diseases of obscure ætiology, a great variety of manifestations of neurasthenia and a host of "other symptoms" and ascribes the cause in all to what he is pleased to call "masked tuberculosis." He then gives us his specific cure, the treatment by "immune blood," which seems to be equally efficacious, whether given by injection or inunction!

Professor Poncet contributes a preface wherein he states simply, but perhaps a little sadly, of Dr. Hollòs's work: "It will astonish many of us, it will shock others, it will convince some." We would confess to all three emotions and would unhesitatingly state our conviction that Dr. Hollòs's work, in the expressive language of his country of adoption, is "all bunk."

Briefly stated, Dr. Hollòs's theory is that most of us have an "hereditary immunity" to tuberculosis and therefore, as nearly everyone becomes infected, there are many patients with "attenuated tuberculosis" which, needless to say, gives "a most varied clinical picture." Apparently all endocrine disturbances are due to tuberculous toxins, so also are psoriasis, epilepsy and most menstrual disorders. Neurasthenics, of course, are too good to let slip. "Especially characteristic is the great feeling of fatigue," worse in the morning, even in "strong, muscular individuals." Headache, vertigo, even blushing come under the ban. What a scope for the unscrupulous therapeutist! "Can we replace the term rheumatism by tuberculosis?" To this preposterous question the author shouts an enthusiastic "yes."

But the most amazing feature of the book are the clinical notes of cases. Some quotations will serve to illustrate the clinical acumen of Dr. Hollòs and his mental outlook on disease. A woman who from her history was obviously suffering from cholelithiasis, came under his care. "The following day, after the second injection, she was taken with a severe gall bladder attack, which was repeated for several days. After the third injection a dyspnœa and a new gall bladder attack appeared. The patient lost faith because of the painful reactions and stopped the treatment." And thereby saved her life no doubt! Less fortunate was this patient who, while undergoing a second course of injections, "vomited for four days. This proved to be a symptom of peptic ulcer of the stomach. The perforation of the ulcer took place and she died a few days later." For brazen effrontery that would appear to be hard to beat, but we can recommend the following: "It was that of a nine year old girl with advanced tuberculosis of the lungs and bowels . . . After the first injection of VIIIth dilution the fever rose. After the second injection the fever rose once more, still higher, and at the height of the reaction the child died." And this is the twentieth century!

We may leave it to New York to decide whether the author of these barefaced admissions of malpraxis is not a menace to the community. Fortunately, it seems hardly possible, outside the United States, that the outrageous nonsense printed in this book, will gain for Dr. Hollòs any followers.

[1] "Tuberculous Intoxications: Concealed and Masked Tuberculosis: A Clinical Study," by Joseph Hollòs, M.D.; 1928. Edinburgh: E. and S. Livingstone. Demy 8vo., pp. 140. Price: 10s. 6d. net.

PROGRESS IN THERAPEUTICS.

"GENERAL THERAPEUTICS," by Professor Bernard Fantus, belongs to "The Practical Medicine Series" which deals with the year's progress in various branches of medicine and surgery.[1] This year (1927) has not been one of therapeutic fireworks. The liver treatment of Addisonian anæmia has become thoroughly stabilized. Reference is made to the correct method of cooking that viscus, as well as various little devices for making more attractive and more palatable what must surely be a monotonous article of dietary. Mercurochrome is a fashionable drug. It has been used in the treatment of pneumonia, septicæmia, genito-urinary infections, furunculosis and as a vaginal antiseptic in obstetrical practice. The American public appears to have abandoned tincture of iodine and taken to mercurochrome as a domestic first aid antiseptic. Reported results are not convincing and the popularity of the remedy is probably due to the shrewd advertising methods on the part of the manufacturing chemists. Bismuth in the treatment of syphilis has had a great vogue. Here again we appear to be on the crest of a fashionable wave. In the process of changing new gods for old we are inclined to forget that mercury has stood the test of time.

More than a third of the book is devoted to physical therapy. Recent work on massage, electrotherapy, hydrotherapy, diathermy, phototherapy and radium therapy have been faithfully and fairly abstracted. Tonsil electrocoagulation is described in full. It sounds suspiciously easy.

This is not a book to buy. Owing to its peculiar arrangement it is doubtful if it has any real function as a book of reference. The printing is good and the index adequate.

[1] "The Practical Medicine Series, Comprising Eight Volumes on the Year's Progress in Medicine and Surgery"; Under the General Editorial Charge of Charles L. Mix, A.M., M.D.; General Therapeutics; 1927. Chicago: The Year Book Publishers. Crown 8vo., pp. 407. Price: $2.25 net.

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The Australasian Medical Publishing Company.

WHEN the Australasian Medical Publishing Company, Limited, was formed some fifteen years ago, the Federal Committee of the British Medical Association in Australia realized that it would be necessary to provide an elastic constitution, in view of the fact that it had no assets and that THE MEDICAL JOURNAL OF AUSTRALIA would have to be developed from nothing. Prices were low in those days, there was a wave of commercial prosperity in Australia and the company had no reason to anticipate any serious obstacle to success. Within two months of the institution of this journal everything was changed. The world became involved in the greatest tragedy of all times. No more unfortunate period could have been selected for the starting of a company that was to provide the medical profession with one federal journal and that might serve the world of science in the Commonwealth in other ways. At first there was no need for any substantial capital. A small sum was raised on debentures to cover the initial expenses contingent on the establishment of the company and the production of the journal. The journal was given fo a commercial printing firm to produce. This firm had not previously handled medical or scientific material and it was unprepared typographically to satisfy the demands of a modern medical periodical. As the years passed and peace beckoned vaguely from the distant future, the difficulties rose in giant form and assumed a threatening aspect. All commodities became costly. The presence of the profiteer was unmistakable in many markets. The price of paper became almost prohibitive and at a later date there was an acute shortage of all classes of printing paper. Exorbitant sums had to be paid for the roughest newsprint. Then came the armistice. The actual slaughter in Europe and in the other theatres of war ceased, but there was no real peace. The allies had won the Great War, but it remained to be seen which nation would emerge least scarred and bruised from the Great Peace. During the early years after the armistice prices soared and industrial unrest further postponed the advent of normal conditions. In 1920 the Australasian Medical Publishing Company, Limited, having survived the disastrous ravages of six terrible years, was faced with a serious dilemma. THE MEDICAL JOURNAL OF AUSTRALIA had to be reduced in size, because of the increasing cost of production. An expedient had to be found to insure the continuation of the journal and to render its expansion possible. The remedy was found. A linotype machine and a miniature composing plant were installed in office rooms in the B.M.A. Building, Elizabeth Street, Sydney, and a staff of two journeymen and one apprentice was engaged. The cost of this installation was met out of income. In the course of three years the company paid off all the instalments without having to approach the medical profession for more money. But there was one defect in the arrangement. The type setting and composing work was in the hands of two expert workers who could not be replaced at short notice in the event of illness. Difficulties were experienced during the annual holiday of each of these workers. In addition the management was also in the hands of one individual. It was recognized that so small an organization was being maintained without any margin of safety and an untoward accident might jeopardize the whole concern. Moreover, the smallness of the printing equipment set a definite limit to the possible expansion of the journal.

The next step was the natural corollary to the first. The constituton of the company permitted the Directors to engage in trade as printers. It would have been economically unsound to have endeavoured to set up a printing plant for the sole purpose of producing the journal. One linotype was scarely sufficient for the type setting, while two were more than was needed. One printing press could have produced much more than five thousand copies of a sixty-four page journal each week. It was therefore determined to build and equip a complete printing house at which the printing of

scientific matter could be carried out by a specially trained staff. For this purpose the medical profession was invited to provide the minimum amount of money required as loans secured by debentures. The company has established in the course of three and a half years a sound business and has gained recognition as accurate and good printers. The period of initiation before a business of this kind can be stabilized is usually from three to four years. Adjustments have had to be made for the peculiarities of a scientific press and difficulties have had to be overcome in connexion with the competition with commercial firms. The year that has just ended, has witnessed a substantial increase in the amount of work completed and for the first time a considerable profit has been earned. The company still has heavy financial obligations, but the future seems to be assured, and the medical profession now possesses a scientific press to which it can turn with confidence. Much hard work will have to be accomplished before the complete success of the undertaking will become apparent. The medical profession has hitherto given some but not extensive support to its press. Medical practitioners are asked to give the company preference when ordering professional stationery. They will receive good paper, well printed at reasonable prices and will have the satisfaction of knowing that they are supporting their own press. The College of Surgeons of Australasia has entrusted the company with the printing of its new journal and this admirable magazine is now available and should be purchased by every practitioner in the Commonwealth who is interested in surgery. There are many other ways in which the support and help of the profession can be extended and we appeal to each member to contribute his share to the success of the undertaking which has the main object of expanding the usefulness of the journal and other publications for the medical profession.

Current Comment.

PROGRESSIVE LENTICULAR DEGENERATION.

SINCE Kinnier Wilson first described the condition known as progressive lenticular degeneration, the relationship between disease of the liver and pathological changes in the tissues of the brain has been the subject of much investigation. In progressive lenticular degeneration there is bilateral degeneration of the lenticular nuclei and this lesion of the central nervous system is invariably accompanied by cirrhosis of the liver. Schütte described a progressive disease of the central nervous system and at autopsy he found that, associated with disease of the liver, there was extensive destruction of nerve cells and fibres together with overgrowth of neuroglia, especially in the frontal lobes. He believed that the hepatic and cerebral lesions were related. Bostroem, in reporting a case of pseudosclerosis, came to the conclusion that the change in the neurone cells and the overgrowth of neuroglia, as well as the alteration in the liver, were caused by intestinal intoxication, dependent on a functional disturbance of the alimentary tract. Disturbance of the protein metabolism arising through lesions of the liver and kidney has been regarded as likely to produce mental symptoms. Weston found a low nitrogen value in manic depressive insanity, in *dementia præcox* and in epilepsy and Frigerio has expressed the opinion that the estimation of non-coagulable nitrogen of the blood is useful in the practice of psychiatry and he regards the symptom of confusion as due to nitrogen retention. Several writers have described hepatic involvement following epidemic encephalitis, but O'Flynn and Critchley were unable to find any clinical, histological or biochemical evidence that the liver is affected. Since the liver plays such an important part in the metabolism of the body, it would not be difficult to understand that a disorder in its function might affect the higher cerebral centres, but it is not at all an easy matter to determine the relationship of liver damage to lenticular degeneration.

E. W. and P. E. Hurst have made an experimental investigation into this relationship.[1] They state that if the relation of the liver disease to the cerebral degeneration is that of cause and effect, the nervous degeneration may be due to the retention of normal metabolic products usually destroyed in or excreted by the liver; it may be due to toxic substances of exogenous origin, allowed by the damaged organ to pass from the portal into the systemic circulation; it may be due to toxic substances produced by abnormal metabolism of the injured liver cells. They state that if the hepatic and cerebral lesions are merely associated in the sense that they are the various effects of a common cause, it is possible to study the conditions experimentally only when the cause is known, as in manganese poisoning. If a factor in the production of the disease is some congenital deficiency in the make-up of the individual, any attempt at experimental reproduction will almost certainly end in failure.

The investigations undertaken by the two authors consisted first of all of a minute examination by modern neurological methods of the nervous systems of five persons dead of cirrhosis of the liver. Four of these suffered from cirrhosis of the Laennec type and in one instance the cirrhosis was accompanied by carcinomatous changes in the areas of regenera-

[1] *The Journal of Pathology and Bacteriology*, April, 1928.

ting liver cells. In none were nervous lesions present other than those found in controls of approximately the same age obtained during routine *post mortem* work. The rest of the experimental work fell into two categories: the production of cirrhosis and the determination of the action of various toxic substances on cirrhotic animals. It will be impossible in the available space to describe the details of all their experiments; if a general outline be given, it will be possible to follow their arguments and discuss their conclusions. Rabbits and guinea pigs were used. When injections of manganese were given, the former were found to be more susceptible than the latter. Although in the rabbit large doses produced severe degeneration in both liver and kidney, in both species smaller and oft repeated doses acted chiefly or entirely on the liver. In rabbits which received relatively smaller doses than guinea pigs, necrosis was a more prominent feature than in the latter animals, it affected a definite proportion of the cells of each lobule and produced a zone of necrotic tissue around each portal space. The appearance of the liver was very similar to that seen in human monolobular cirrhosis. In both groups of animals the usual concomitants of cirrhosis, such as proliferation of small bile ducts, fatty degeneration and so forth, were present. In neither species were any changes found in the central nervous system. A suspension of *Bacillus coli* was injected from time to time into one of the heart cavities of guinea pigs. It was found that in the earlier stages necrosis was much more pronounced and later on the process of fibrosis was greatly stimulated, so that an intense cirrhosis was produced in a comparatively short time. The cirrhosis resembled some of the more acute forms of human cirrhosis. Chloroform was injected since it was thought that its action on the central regions of the liver lobules might enhance the effects of manganese on the peripheral parts of the lobules. The results were inconclusive and the fibrous tissue produced was only slightly, if at all, more than that produced by manganese alone. Phenylhydrazine was also used because it has a destructive effect on red blood corpuscles and because it exerts a toxic effect on the liver. Fatty degeneration of the liver resulted and central cells of the lobules were affected by hydropic distension. It rarely caused cirrhosis to appear. When, however, it was used with manganese, it augmented the effect of the latter. The injection of bile did not give rise to cirrhosis. None of these poisons produced any degenerative changes in the central nervous system. With guanidin the reverse conditions resulted: no effect was produced on the liver, but severe degeneration of the nerve cells was found with amœboid changes in the neuroglia.

E. W. and P. E. Hurst discuss the production of experimental cirrhosis at some length, but their remarks on the influence of liver disease on the central nervous system are disappointing. They state that none of the substances or combinations of substances used in the production of cirrhosis gave rise to any histological changes which could be accepted as a positive result, and that when slight degenerative phenomena in the nerve cells were found, they were such as might have occurred at or immediately before death as a terminal phenomenon due to extraneous factors. In their opinion the results of their guanidin experiments on animals with a damaged liver merely show that once the poisonous threshold for that body is passed, a fatal termination occurs with greater frequency than in a healthy animal; the threshold is not appreciably lowered by the liver disease, nor are the histological changes in the central nervous system more intense. Their general conclusion is that they obtained no evidence of any connexion between liver cirrhosis and cerebral changes which might throw light on what they call hepato-lenticular degeneration.

This is not surprising, for there appears to be no evidence to suggest that the cause of progressive lenticular degeneration acts in a manner similar to a heavy metal. Manganese like other heavy metals has an action which is dependent on the sequence of its effects. It affects nerve cells, liver cells, blood cells and kidney cells in this order. In his original contribution Wilson states that progressive lenticular degeneration is not due to a congenital or abiotrophic defect, the presumption being strong that the disease is acquired. He found evidence that the disease is toxic in nature, but none to suggest that the toxin is syphilitic. He thought it possible that the toxin was elaborated in the liver and found that the toxin has a specific action on the lenticular neucleus. He stated that the nature of the toxin was speculative and that it was almost certainly not microbial. The familial nature of the disease also needs to be emphasized. The work of E. W. and P. E. Hurst is concerned with a hypothetical ætiology which may or may not be its true explanation. It is of interest to draw attention to the similarity of this disease to pernicious anæmia. In both diseases there is a disturbance of the function and/or structure of the liver and definite and peculiar nerve changes occur. If this similarity is borne in mind by investigators useful results may be obtained. At the present time, there is nothing to be added to Wilson's original description.

THE COMBATING OF VENEREAL DISEASES.

WE have been informed that it is proposed to institute a society for the prevention of venereal diseases in New South Wales. The organizers wish to provide young persons with accurate information regarding venereal diseases, to educate medical students and general practitioners in the modern methods of diagnosis and treatment, to publish a journal of social hygiene in which the names and addresses of all members of the British Medical Association who practise as "venereologists" shall appear, to authorize and advertise the sale of a prophylactic cream of approved potency and efficiency and to advocate reforms. It would be interesting to learn the views of the Medical Board on these proposals.

Abstracts from Current Medical Literature.

OPHTHALMOLOGY.

Optical Iridectomy in Congenital Cataract.

L. WEBSTER FOX (*The Journal of the American Medical Association*, December, 1927) discusses the operative treatment of congenital cataract. He describes the unsatisfactory treatment by numerous needlings and states that it often results in poor vision and the necessity for the use of high convex glasses. If the periphery of both lenses is clear, the most suitable operation is a small optical iridectomy at the nasal side in each eye, performed under general anæsthesia and placed to permit binocular vision. This procedure is sometimes inapplicable, but since adopting this method the author is surprised at its wide range of application. Should the lenses subsequently need removal, the iridectomy is useful and suitably placed.

Pseudoglaucoma.

A. FUCHS (*British Journal of Ophthalmology*, February, 1928) reports several cases of cupping of the optic disc which simulated glaucomatous cupping. In the right eye of a woman of sixty-four there was complete excavation of the disc of four diopters. In the nasal part of the cup there was a sharply defined pit. The tension was normal. In another woman, aged forty-three, there was deep excavation of the right disc, the floor being yellowish-white and covered by semi-opaque tissue obscuring the vessels. The right papilla was larger than the left. The condition was one of congenital colobomatous excavation simulating *glaucoma simplex*. A similar disc was seen in a girl of twelve and a half years. Angiectasia of the eyeball resembles inflammatory glaucoma. A boy, aged fourteen years, from Cairo, had his eyes injected for five years. A well known oculist advised operation for glaucoma. On the eyeball were seen numerous vessels of unusual breadth and tortuosity running from the limbus backwards. The lids and fornices were also hyperæmic. Otherwise the eyes were normal. Leber has reported two similar cases.

Treatment of Cataract with Lens Antigen.

Z. H. ELLIS (*Archives of Ophthalmology*, January, 1928) treated a series of patients with incipient cataract by injections of lens antigen in the manner described by A. E. Davis. The patients were instructed to be abstemious in the use of tobacco and alcohol and were given potassium iodide three times a day. The reaction was tested by the intracutaneous injection of 0·06 mil (one minim) of lens antigen. Subcutaneous injections of half a cubic centimetre of antigen were then given. The following day one cubic centimetre was given and the amount was increased by one cubic centimetre per day until the dose was ten cubic centimetres. Another series of patients was watched without any treatment. It was found that the condition of many of these remained stationary and the vision varied. In the twenty-seven cataracts treated with lens antigen the results were: Progress of the cataract, 14; no change, 13; absorption of cataract, as shown by the slit lamp or by improvement in vision, none. One exhibited anaphylactic shock after the thirtieth injection. Most of the patients noted a general tonic effect from the lens protein.

Syphilitic Ocular Lesions.

K. LINDNER (*Wiener Medizinische Wochenschrift*, May 5, 1928) describes the principal ocular lesions caused by syphilis. Primary affections are very rare. The danger of acute iritis in the secondary stage lies in the risk of adhesions between the iris and lens. A positive response to the Wassermann test does not absolutely exclude tuberculosis in cases of subacute iritis. Gumma of the orbit is frequently mistaken for tumour formation, despite the Wassermann reaction or even exploratory incision. The Argyll-Robertson pupil may be present only in one eye and further development may be slow. Tabetic affections of the ocular muscles frequently manifest remissions, but any pupillary changes are permanent. Optic atrophy is generally due to the effect of toxins on the optic nerve and not to a direct influence of the spirochætes on the nerve substance. The onset of optic atrophy is generally insidious and the diminution in the field of vision may not be noted until atrophy is far advanced. The result of treatment is very disappointing. The combined malaria-"Salvarsan" cure is dangerous. The only hope is to treat tabetic symptoms at an early stage, for in advanced cases little improvement can be expected. In congenital conditions the iritis present is of the chronic type. *Keratitis parenchymatosa* is very resistant to any treatment. In spite of this treatment may hinder tabes or optic atrophy at a later date. However, in many instances corneal ulceration eventually leads to blindness. Formerly congenital syphilis was diagnosed by the triad of lesions of the eyes, ears and teeth. Nowadays the only symptom is frequently a *keratitis parenchymatosa*.

Angioid Streaks of the Fundus Oculi.

F. P. CALHOUN (*American Journal of Ophthalmology*, February, 1928) reports a case in which angioid streaks were seen in the *fundus oculi* and reviews the literature of the condition. The streaks are situated deeper than the retinal vessels and the lines have a circular arrangement. A short distance from the disc margin and from this circle other lines radiate and anastomose with one another. Sometimes white lines follow the streak and areas of chorioidal atrophy are seen. Collins has an anatomical explanation to offer. The short posterior ciliary arteries penetrate the sclera and form a ring, the circle of Zinn, a short distance around the nerve margin; from this circle branches extend forward to supply the posterior half of the chorioid. Subchorioidal hæmorrhages in this stagnant region are badly absorbed; hæmoglobin crystals tend to break up and become deposited along the path of the arteries.

Retroconjunctival Posterior Sclerotomy.

J. S. FRIEDENWALD (*American Journal of Ophthalmology*, February, 1928) has devised an operation of retroconjunctival posterior sclerotomy for the relief of glaucomatous tension when the situation is complicated by corneal or conjunctival sepsis. The method consists of making the incision through the skin near the lower temporal angle of the orbit. The conjunctiva of the fornix is held out of the way with fixation forceps, while the knife is directed towards the equator of the eyeball in the lower temporal quadrant.

Operation for Ptosis.

G. S. DERBY (*American Journal of Ophthalmology*, May, 1928) suggests the use of a strip of *fascia lata* in operating for ptosis. A skin incision is made just above the lashes and the upper edge is dissected back for a short distance. Two tunnels are made under the skin to points above the eyebrow by means of a double-edged pointed knife. The projecting blade of the knife is lightly grasped with a mosquito forceps and, as the knife is withdrawn, the forceps follow through the tunnel and emerge at the lid margin. A piece of *fascia lata*, seven to nine centimetres long and one centimetre wide, is then secured and each end is grasped by the forceps and drawn up to emerge above the eyebrow. One end is then sutured to the *frontalis* as close to the periosteum as possible. The other free end is then drawn up to the required degree of tension and fixed.

Detachment of the Zonula Lamella.

S. J. MEYER (*Archives of Ophthalmology*, March, 1928) refers to Elschnig's findings in 1922 concerning the eyes of two glass blowers, namely: "on the anterior lens surface there lies a polygonal, thin transparent membrane with gold glistening margins which vibrated upon any eye movement. The margins were rolled lightly anteriorly, especially above, so that the entire formation appears to be attached to the lens only in its middle part and extends concavely towards the anterior chamber." The *zonula lamella* is the most anterior part of the lens capsule. The author reports two cases. The first patient was a blacksmith of forty-eight years, blind in the left eye and with poor sight in the right. Slit-lamp examination of the right eye revealed the *zonula lamella* detached from the lens

capsule on all sides except the temporal part. The edges curl forward. The second patient, a woman of fifty-five, a luetic subject, had an intracapsular extraction performed in the left eye. Slit-lamp examination after the extraction revealed a glistening membrane moderately thick and pigmented extending forward into the anterior chamber. This can be nothing but the *zanula lamella* still attached to the zonular fibres at the temporal margin.

LARYNGOLOGY AND OTOLOGY.

Nasal Polypi and Inflammation of the Accessory Sinuses of the Nose.

T. B. LAYTON (*Journal of Laryngology and Otology*, November, 1927) agrees with the view that nasal polypi are inflammatory in origin and not benign tumours as formerly thought. Their exact nature and meaning are still unknown. Authorities differ as to the presence of underlying sinus disease. The author considers that there is sinus disease and that simple removal of polypi never cures except when the polypus is single and growing from a pedicle beneath the inferior turbinate bone. Sinus disease may be acute, chronic suppurative or non-suppurative (catarrhal). Chronic suppurative and chronic catarrhal sinusitis run separate courses and do not pass the one into the other. Polypi do not accompany suppurative sinusitis and chronic catarrhal sinusitis is not the result of suppurative sinusitis or *vice versa*. Although muco-pus is seen in the nose in the presence of polypi, careful examination by the author has never revealed a suppurative condition. The treatment adopted is the complete removal of the ethmoid area along with the polypi and for this the author now prefers an external approach through the modified incision used for draining the frontal sinus.

Indications for Tonsillectomy.

M. HAJEK (*Wiener Medizinische Wochenschrift*, January 21, 1928) states that an absolute indication for tonsillectomy is the recurrence of anginal attacks with or without local complications such as peritonsillar abscess. Angina associated with rheumatism, endocarditis and nephritis is an example of a relative indication for the operation. Examination of the tonsil does not necessarily afford proof of the need for removal, because in healthy persons secretions may be expressed from the tonsillar crypts. When in spite of the absence of a history of anginal attacks the tonsil is suspected, the patient should be observed for some time and the temperature regularly taken. Many such patients manifest a slight rise of temperature associated with reddening of the tonsil and the expression of more secretion than usual. If this fails, then massage of the tonsil should be performed. There may be a rise of temperature showing that some absorption of pus has occurred. The sound may also be employed to detect small calculi and pockets of pus. The author has not seen any ill effects following tonsillectomy performed during the acute stage with peritonsillar abscess formation.

Surgical Diathermy and Malignant Tumours in the Anterior Air Passages.

E. SCHMIEGELOW (*Laryngoscope*, December, 1927) gives his experiences in the use of surgical diathermy for malignant tumours in the anterior air passages. He believes that this method of destruction is the safest to adopt in growths occurring in all areas of the nose, accessory sinuses, mouth, jaws and throat. In very extensive growths much relief can be given and life prolonged by this treatment, even if complete removal is impossible. Radical destruction of all the affected area, bone included, is adopted. For instance, in cancer of the upper jaw the usual skin incision is made and in addition to the whole growth the palate is destroyed. This throws the nasal cavities into one and allows of free inspection of the whole area at any time to detect recurrence. A dental plate is found satisfactory in covering the perforation. The treatment must be thorough and consists of coagulating and then removing the coagulum with a sharp spoon until all areas are dealt with. The author frequently spends two to three hours in obtaining these results. Shock is slight and healing clean and rapid. In naso-pharyngeal growths visual access by dealing with the soft palate first is recommended. In treating neoplasms of the larynx the author has not experienced the untoward symptoms reported by other surgeons. A unilateral early intrinsic cancer of the larynx is best treated by thyreofissure and excision, but more extensive growths can be effectively dealt with by diathermy and better results obtained than by total laryngectomy. Further, the treatment is easier for both surgeon and patient and causes less shock and quicker convalescence. Preliminary tracheotomy is advisable in very debilitated patients; usually suspension laryngoscopy under deep chloroform narcosis is adopted.

The Use of Barbital with Local Anæsthetics.

C. B. WILLIAMS (*Laryngoscope*, December, 1927) reports successes with the administration of barbital in preventing the toxicosis from local anæsthetics. Tatum, Atkinson and Collins have demonstrated that intravenous injections of sodium barbital and paraldehyde increase the cocaine tolerance of dogs by 400% and Leshure reported favourably on the use of barbital by the mouth in a series of one hundred patients. The author prescribes 0·3 gramme (five grains) of barbital two hours before operation, the dose is repeated one hour later. It has been found that the nervous stability of patients has been increased and toxic manifestations have been prevented.

Hæmorrhage After Tonsillectomy.

ALBERT T. TIBBETS (*Laryngoscope*, December, 1927) gives a report of five cases in which hæmorrhage occurred after operation of enucleation of tonsils. In each instance swabbings revealed the presence of the organisms of Vincent's angina. All the patients were adults, three being males and two females. There was a history in each of recurrent tonsillitis but none of Vincent's angina infection. Four had general anæsthesia, one local. Hæmorrhage occurred five hours after operation in one patient and in the others from two to seven days later. Specific treatment for Vincent's angina quickly brought about cessation of the hæmorrhage.

Ionization in the Nose.

ANDREW CAMPBELL (*Journal of Laryngology and Otology*, February, 1928) publishes a new technique in treating the nasal cavities by ionization. It is essential to adopt Freil's advice "to fill the affected cavity and to keep it full," but the author found Freil's technique quite impossible and painful in practice. In the author's method the patient lies face downwards with head and neck extending over the end of the couch, the forehead is supported by resting it on a stool placed at the head of the couch. The nostril of the affected side is plugged with vaseline-smeared plasticine and a small insulated cannula is pushed through the plug so that its uninsulated free end lies just beyond the nasal vestibule. The cannula is connected by a tube to a douche can and the active electrode is also attached to it. The ionizing fluid is then run slowly into the nose until it is returned through the other nostril over the free posterior border of the nasal septum. This indicates that all sinuses on this side are full of fluid, except the sphenoidal sinus. The current is then turned on and the lotion kept dripping slowly during the whole treatment. This procedure is found to be very simple and painless in practice and to give excellent results. The author found it to be most effective in post-operative treatment of sinusitis when chlorine is the active ionizing element used. Zinc is of benefit after the removal of nasal polypi and bismuth has lately been used in hay fever. The results in atrophic rhinitis have been disappointing.

Foreign Body in the Œsophagus Causing Dyspnœa.

W. S. THACKER NEVILLE (*Journal of Laryngology and Otology*, February, 1928) describes a case in which a foreign body in the œsophagus caused dyspnœa and huskiness. The foreign body, a large piece of steak, was strongly held by the *crico-pharyngeus* muscle and its friability made extraction difficult. The author used a new modification of Jackson's laryngoscope. The tube is divided into two halves, an anterior and posterior. These two halves can be screwed open by a ratchet screw and the author considers that the extra room for manipulation thus gained is a great advantage.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION in conjunction with the Section of Obstetrics and Gynæcology was held at the B.M.A. Building, 30-34, Elizabeth Street, Sydney, on June 28, 1928, DR. J. E. V. BARLING, the President, in the chair.

Menorrhagia, Uterine Inertia.

DR. J. L. T. ISBISTER read a paper entitled: "Menorrhagia with an Apparently Normal Pelvis" (see THE MEDICAL JOURNAL OF AUSTRALIA, August 11, 1928, page 165).

DR. A. J. GIBSON read a paper entitled: "Faults in the Uterine Powers" (see THE MEDICAL JOURNAL OF AUSTRALIA, August 11, 1928, page 168).

PROFESSOR J. C. WINDEYER, in discussing Dr. Isbister's paper, said that the main point that had been brought out was that the curette should not be used in all cases of menorrhagia, but should be reserved for special conditions. All possibilities should be taken into consideration. Dr. Gibson had dealt with the abnormalities of the uterine powers in only the first and second stages of labour. He was disappointed that he had not mentioned the third stage, but presumed that this omission was necessary on account of an undue lengthening of the paper. He agreed that change of posture often worked wonders. There were two or three points which had emerged during recent years in regard to treatment. The first was that extract of pituitary gland had been standardized. Ten units were contained in every cubic centimetre. Formerly pituitary extract had been of indefinite strength and had therefore, been unreliable. Burn and Bourne had shown that two units of the standardized extract constituted the maximum safe dose. In the past pituitrin had caused maternal and infantile deaths and much maternal trauma. At the Johns Hopkins Hospital the nasal method of administration had been used. Advantages had been claimed for this method, that as the wool could be withdrawn at any time, a continued overdose could be avoided. He also pointed out that the liquid extract or ergot of the British Pharmacopœia was an absolutely inert drug both in the third stage and during the puerperium. There were other preparations of ergot that could be used with benefit.

DR. T. W. LIPSCOMB, in referring to Dr. Isbister's paper, said that in young unmarried women the causes of menorrhagia were usually constitutional and that the proper treatment was the administration of calcium lactate and thyreoid extract in small doses. In married women, however, it was more likely that a pelvic cause could be found. He thought that it was as well to lay stress on the frequent unnecessary use of the curette in this connexion. He had no liking for radiation in the treatment of uterine hæmorrhage. Radiologists held that adnexal disease was a contraindication to the application of X rays and he defied anyone to tell with certainty whether adnexal disease was present or not.

DR. F. A. MAGUIRE emphasized the medical aspect of the subject of menorrhagia especially in young women. He held strongly that all the systems of the body should be thoroughly investigated. Menorrhagia in young girls was usually controllable by means of calcium lactate. This bore out Blair Bell's teaching that it was the function of the uterus to pick out calcium from the blood. Blair Bell had shown that the uterus used this function for the purpose of supplying the fœtus with calcium for its growing bones. It was necessary to exclude conditions such as purpura in forming a diagnosis. He referred to a case to illustrate this point. In many young girls the chain of the glands of internal secretion became disturbed. There was either over or under action of the thyreoid gland. The menstrual flow varied in inverse proportion to the activity of the thyreoid gland. In regard to the musculature of the uterus he recalled the fact that during pregnancy the round ligaments became greatly hypertrophied and performed their functions as powerful guy ropes.

He had recently read the record of a discussion which had taken place in the Royal Society of Medicine on the use of anæsthesia in labour. Chloral was advocated for the first stage and chloroform for the second. Chloroform had the power of overcoming the resistance of the *levatores ani* to the uterus and the muscles of the abdominal wall.

DR. C. V. BOWKER expressed his indebtedness to the readers of the papers and said that the present trend was towards thorough investigation and diagnosis before extreme measures such as hysterectomy were undertaken. He referred particularly to the part played by *fibrosis uteri* especially when no abnormal conditions would be found in the pelvis. In many instances this state of affairs could be explained by ovarian dysfunction. Attempts had been made to advance the knowledge of the use of X rays and radium in these conditions of uterine hæmorrhage and this work could not be ignored, for any benign conditions could be successfully treated by this agency. X rays and radium, however, had to be used with discretion and at present were not adaptable to young adults for fear of permanent destruction of ovarian function. It was true that one or two Continental gynæcologists had determined the exact doses which sufficed to produce a temporary amenorrhœa, but the line of demarcation was doubtful and care had to be exercised. Many had found that when radium was used in patients over forty years of age the after effects were severer than when it was used in the treatment of impaired function in younger people.

DR. H. A. RIDLER pointed out that in hyperthyreoidism there was a very considerable variation in the effect on the menstrual function. Returning to the question of posture he agreed with what had been said, but he pointed out that at times benefit resulted from the change from the dorsal to the lateral position. He asked those present if they could make any suggestions with regard to *multiparæ* whose membranes ruptured from the sixth to eighth and a half month of pregnancy and who failed to come into labour. His experience had been that when ordinary methods of induction, namely, quinine and castor oil, bougies, Champetier de Ribes's bag or podalic version, failed to induce labour within a reasonable time, the condition ended fatally, death being due to sepsis.

DR. N. D. ROYLE dealt with the nerve supply of the uterus. He pointed out that this supply was derived from the sacral as well as the sympathetic nerve ganglia. The stimulation of the sympathetic nerves by adrenalin inhibited the contraction of the uterine muscle and was therefore antagonistic to the action of the sacral nerves. He thought that it was wrong to use the term "tonic" contractions of the uterine muscle. The sacral nerve was a motor nerve and over-stimulation of a motor nerve merely gave rise to sustained contraction. When "Stovaine" was injected into the lumbar region of the spinal theca, it was impossible to say how far the effect extended and probably both the sympathetic and the sacral nerves were paralysed. In other words the uterus was denervated. In inertia there was over-action of the sympathetic nervous system. Dr. Gibson's method of dealing with this condition in which he administered hot enemata in order to produce contraction of the uterus was really causing inhibition of the sympathetic nerve supply. It would be interesting to see the effect of diathermy on inertia of the uterus.

In reply to Dr. Maguire, Dr. Royle stated he did not know whether "Stovaine" would render Cæsarean section a bloodless operation. Presumably it would paralyse the vaso constrictors of the blood vessels as well as the uterine muscles and cause a low blood pressure, but he was not prepared to express an opinion as to its effect on bleeding.

DR. J. CRAWFORD ROBERTSON pointed out that the speakers had given an admirable summary of the present position of the knowledge of the subjects under discussion and had dealt with a great deal of the work done by Whitehouse, more especially in regard to the function of the ovaries. In abdominal section surgeons usually paid very little attention to the condition of the ovary. Examination was casual and insufficient attention was paid to any cystic condition. Particular care should also be taken in salpingectomy not to interfere in any way with the circulation of the ovary, otherwise a cystic condition was likely to be produced. In dealing with the menorrhagia

of puberty he stated that this was frequently dependent on developmental changes. Extract of the anterior portion of the pituitary gland often did good. He was sanguine that improvements would be made in connexion with the extract of ovary substance. This extract should be given intramuscularly, but not by mouth.

DR. ISBISTER in his reply stated that hæmorrhage might be of ovarian or constitutional origin. It was easy to overlook or mistake the signs of these conditions. He dealt with cases of fatal hæmorrhage and referred to the work of Graves, of Boston. In regard to thyreoid disease, according to the modern view, it was quite essential to estimate the basal metabolic rate. By means of Reid's formula this was easily done. In the formula there were two constants. The formula was:

$$B.M.R. = 0{\cdot}7\,[P + 0{\cdot}9\,(S - D)] - 71{\cdot}5$$

P represented the pulse rate, S systolic blood pressure and D the diastolic blood pressure. It was stated that the figure arrived at in this way was substantially correct. Finally in dealing with radiation Dr. Isbister thought that it was risky to make any definite statements. There was a necessity to exercise care in the selection of patients as its effect on the ovary was not too certainly known. They would be treading on dangerous ground if without due consideration they applied radiation to young women. He had seen peritonitis follow on the use of radium.

DR. GIBSON in his reply spoke of the effect of chloroform in the first stage and referred to a patient with a rigid *os* to whom it had been given with successful results. In regard to the question of posture he held that it was better to keep the patient on her back until the last phase of the second stage when she could be turned on her left side with advantage. He agreed that the British Pharmacopœia liquid extract of ergot was valueless. The alkaloids of ergot were histamine, ergotoxin and tyramine. Ergotoxin was the only active principle. The British Pharmacopœia extract contained none. An experiment had been tried by comparing the action of the British Pharmacopœia and the United States Pharmacopœia liquid extract of ergot with that of "Marmite." The uterus had involuted equally well with all three. He agreed with Dr. Maguire concerning the action of chloroform in controlling the antagonistic action of the *levatores ani* and the abdominal wall. He thought that there was a tendency on the part of many obstetricians to give too much chloroform. He gave a few drops only during the pains. The patients were grateful for the comfort and it shortened the second stage. In reply to Dr. Ridler he stated that Polak advocated packing round the cervix with wool and glycerine or boroglyceride in dealing with patients in whom the membranes had ruptured prematurely.

MEDICO-POLITICAL.

MEETING OF THE FEDERAL COMMITTEE.

A MEETING OF THE FEDERAL COMMITTEE OF THE BRITISH MEDICAL ASSOCIATION IN AUSTRALIA was held on August 7 and 8, 1928, at the B.M.A. Building, 30-34, Elizabeth Street, Sydney, DR. W. N. ROBERTSON, C.B.E., the Vice-Chairman, in the chair.

Representatives.

The following representatives of the Branches were present:

New South Wales Branch: Dr. R. H. Todd, Dr. J. Adam Dick, C.M.G.
Victorian Branch: Dr. R. H. Fetherston, Dr. F. L. Davies.
Queensland Branch: Dr. W. N. Robertson, C.B.E., Dr. E. Sandford Jackson.
South Australian Branch: Sir Henry Newland, D.S.O., Dr. Bronte Smeaton.
Tasmanian Branch: Dr. Gregory Sprott.

Apologies for non-attendance were received from Dr. F. A. Hadley and Dr. D. Paton, representing the Western Australian Branch, and from Dr. E. Brettingham Moore, representing the Tasmanian Branch.

Sir Henry Newland was appointed proxy for Dr. F. A. Hadley, Dr. R. H. Todd was appointed proxy for Dr. D. D. Paton and Dr. Gregory Sprott was appointed proxy for Dr. Brettingham Moore.

Congratulations to Sir Henry Newland.

DR. W. N. ROBERTSON, the Chairman, asked the members of the Committee to join him in congratulating Sir Henry Newland on having received the honour of knighthood. They all realized that this honour had been richly deserved; it had been earned.

SIR HENRY NEWLAND thanked his colleagues for their kind congratulations. He was proud of the honour that had been bestowed on him, but recognized that it was as much a compliment to the medical profession as to himself.

Financial Statements.

The financial statements in connexion with the Federal Committee and with the Australasian Medical Congress (British Medical Association) Accumulated Funds Account were received and adopted. The statements are published below.

Medical Officers' Relief Fund.

The Honorary Secretary read the report for the Trustees of the Medical Officers' Relief Fund and presented a statement of account. The report and balance sheet were received.

Correspondence.

Letters were read from the Medical Secretary of the British Medical Association and from the Honorary Secretary of the Federal Committee in reply relative to the visit of Sir George Syme to England.

A communication was read from the Public Medical Officers' Association of New South Wales directing the attention of the Federal Committee to the proposal of the Greater Brisbane City Council to abolish the office of medical officer of health for Brisbane. It was noted that the Queensland Branch was considering this matter.

National Insurance.

A discussion took place on the attitude to be adopted by the medical profession in connexion with national in-

FEDERAL COMMITTEE OF THE BRITISH MEDICAL ASSOCIATION IN AUSTRALIA.

STATEMENT OF RECEIPTS AND PAYMENTS FOR SIX MONTHS ENDED JUNE 30, 1928.

Receipts.

	£ s. d.	£ s. d.
To Balance at December 31, 1927—		
Government Savings Bank of New South Wales	544 3 5	
Union Bank of Australia, Limited	324 13 8	
Cash on hand	1 15 1	
		870 12 2
„ Branch Contributions		38 18 2
„ Interest—Government Savings Bank of New South Wales		24 0 3
		£933 10 7

Payments.

	£ s. d.	£ s. d.
By Travelling Expenses..		69 11 10
„ Sundry Expenses		102 10 7
„ Balance at June 30, 1928—		
Government Savings Bank of New South Wales	568 3 8	
Union Bank of Australia, Limited	190 10 6	
Cash on hand	2 14 0	
		761 8 2
		33 10 7

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

ACCUMULATED FUNDS ACCOUNT.

Receipts.	£	s.	d.	*Payments.*	£	s.	d.
To Balance at December 31, 1927—Government Savings Bank of New South Wales ..	373	12	10	By Sundry Expenses—Clerical Assistance	7	10	0
„ Interest—Government Savings Bank of New South Wales	11	6	11	„ Balance at June 30, 1928—Government Savings Bank of New South Wales	377	9	9
	£384	19	9		£384	19	9

(Signed) ROBT. H. TODD,
Honorary Secretary.
Sydney, August 3, 1928.

Audited and found correct.
(Signed) W. H. CRAGO,
Honorary Auditor.

MEDICAL OFFICERS' RELEIF FUND (FEDERAL).

STATEMENT OF CURRENT ACCOUNT FOR THE YEAR ENDED JUNE 30, 1928.

Receipts.	£	s.	d.	*Disbursements.*	£	s.	d.
To Balance forward	355	7	7	By Loans Granted	1,592	2	0
„ Loans Repaid	1,880	10	0	„ Deposit at Call (Earning 5%)	900	0	0
„ Interest Received	461	12	0	„ Benefactions	252	5	0
„ Exchange Added	0	13	4	„ Audit Fee	12	12	0
„ Debtor Balance	62	1	1	„ Bank Charges	3	5	0
	£2,760	4	0		£2,760	4	0

surance which it was understood would soon be introduced. Dr. R. H. Fetherston expressed the opinion that the medical profession should register its determination that there should be no control by friendly societies or by other societies whose ordinary function was to provide medical benefit. It was understood that medical benefit would not be included in any measure that might be introduced in the near future, but it was thought that if the administration were placed in the hands of the friendly societies, it would be extremely difficult for the medical profession to induce the Government to alter this arrangement at a later date if medical benefit were introduced. Dr. Fetherston thought that the friendly societies and similar organizations would be able to harass the medical profession in many ways even under the restricted scheme. Dispensaries and medical institutions might be created to the detriment of the medical profession as a whole. He moved:

> That this Committee is opposed to the benefits under national insurance being administered by approved societies and is of the opinion that the benefits of the proposed act should be administered by the Federal Government, as it was understood would be the case from the discussions with Senator Millen.

DR. F. L. DAVIES seconded the motion. He thought that if the friendly societies obtained control of the sickness benefit, the whole principle of free choice of doctor for certification purposes would be endangered. The friendly societies would find that it was cheaper to engage medical officers at a salary or to guarantee the income of certain medical officers than to allow insured persons to obtain certificates from any medical practitioner prepared to give this service.

It was pointed out that all the Branches had declared their opposition to the recognition of approved societies in national health insurance. Dr. Fetherston's motion was adopted.

DR. F. L. DAVIES suggested that the income limit for sickness benefit under the national insurance scheme should not exceed the existing lodge income limit. The motion was seconded by DR. R. H. FETHERSTON.

After some discussion it was resolved that in the opinion of the Federal Committee the income limit should not exceed £416.

British Medical Association.

In further reference to the matter of the membership of Branches dealt with at the last meeting of the Federal Committee, the Honorary Secretary reported that a reply had been received from the Medical Secretary of the British Medical Association to the effect that the Council of the Association was not prepared to amend Article 17 of the Articles of Association to enable a Council of an overseas Branch to exercise its discretion in accepting a member of the Association as a member of that Branch when he moved from the area of another Branch. The reasons for this decision were set forth in detail in the letter. The letter was received.

The Honorary Secretary reported that copies of the model rules governing procedure in ethical matters had been forwarded to all the Branches (see THE MEDICAL JOURNAL OF AUSTRALIA, May 5, 1928, page 564). Information had been received to the effect that the Queensland Branch had adopted these rules.

Australasian Medical Congress.

It was reported that the Executive Committee of the third session of the Australasian Medical Congress (British Medical Association) had under consideration the question of the date of the session. It had been found that there were relatively few days in August when all medical schools were simultaneously in vacation. It had been suggested that the session be held in January, 1930, when all the medical schools would be in vacation. The selection of this date, however, did not meet with universal favour. It was noted that the matter would be decided by the Executive Committee at an early date.

Federal Registration of Medical Practitioners.

Reference was made to the proposal that medical registration should be undertaken by the Federal Government and that the State Governments should relinquish their rights in connexion with this matter. The matter was in the hands of the State Governments.

Naval Medical Officers.

In accordance with the resolution passed at the last meeting of the Federal Committee (see THE MEDICAL JOURNAL OF AUSTRALIA, May 5, 1928, page 565), Sir George Syme and Dr. R. H. Fetherston had considered as a sub-committee the conditions of service of naval medical officers and had reported as follows:

It has been learnt that the following are subjects of dissatisfaction:

1. Amalgamation of medical services with those of the Australian Army Medical Corps and Royal Australian Air Force Medical Service. This is strongly opposed by all medical and dental officers.

2. Pay of Director of Naval Medical Services. At present the Acting Director of Naval Medical Services with rank of Surgeon-Commander is in a less favourable financial position than his next most senior officers of the same rank who have extra charge pay. Previous holders of the position have held the rank without the pay of a Surgeon-Captain.

3. Retiring age. As there is no provision for the rank of more than one Surgeon-Captain with a retiring age of fifty-five, all medical officers must retire at fifty. There are three Engineer Captains with only one year's more seniority than the three senior surgeons of the Royal Australian Navy.

4. Status of Director of Naval Medical Services. He has no right of access to the Naval Board or Minister of Defence as has the Director of Government Medical Services. His recommendations concerning medical matters and disposition of officers can be vetoed by laymen without any reason being given and he has no opportunity of personally urging his point of view.

5. Deferred pay. In addition to the Naval medical officers who joined prior to the amendment of the Naval Financial Regulation in May, 1923, making deferred pay payable only on final discharge, there are medical officers who have joined since under the impression that their deferred pay would be paid on resignation. It should be further urged that the conditions of service supplied to applicants for commissions in the future should give full and definite information as to the conditions under which deferred pay will be paid. It is thought, however, that the knowledge of the existence of such a regulation concerning the payment of deferred pay would limit considerably the number of applicants for commissions.

6. Pay generally. Active pay about 5s. 6d. per day better than Royal Navy.

(*a*) Increased allowance for specialists; 2s. 6d. per day is allowed in lieu of 5s. in Royal Navy.

(*b*) Charge pay to be granted. There are two hospitals in Australia which in the Royal Navy would carry 10s. per day in the case of a surgeon-captain or 5s. in the case of a surgeon-commander.

(*c*) No widows' pensions granted as in Royal Navy.

(*d*) No married officers quarters provided as in Royal Navy.

(*e*) Retiring payment much lower than in Royal Navy. Example: After twenty years Royal Australian Navy gets £3,710. After twenty years Royal Navy gets £3,113 + £251 *per annum* = £6,227.

It was resolved to adopt the report and to forward it to the Minister for Defence with a request that effect be given to the recommendations contained therein. It was further determined to address a copy of the report together with an accompanying letter to the Director of Naval Medical Services.

Travelling Medical Examiners for Life Insurance Companies.

At its meeting in April, 1928, the Federal Committee determined that four resolutions passed at that meeting should be communicated to the Life Offices' Association of Australia. Some difficulty had been experienced in regard to the third resolution in the following effect:

> That travelling allowances be paid including hotel and other maintenance expenses.

Further inquiry had elicited the fact that the majority of life insurance societies paid small fees for services and totally insufficient amounts as maintenance allowances in addition to travelling expenses. The lowest maintenance allowance paid was ten shillings and sixpence and the highest one pound. It was resolved that a minimum amount of twenty-five shillings a day as maintenance allowance be asked for.

Government Appointments.

A letter was read from the Victorian Branch suggesting that all vacancies for government appointments should be advertised in both the daily and the medical press. This communication had been referred to the Branches. A reply had been received from the Western Australian Branch approving of the suggestion. In the course of discussion it was pointed out that some difficulty was experienced in connexion with the definition of a government appointment. It was at times not clear whether an appointment was or was not a government one. An example of this was cited in connexion with medical officers to baby health centres. Dr. R. H. Fetherston reminded the members that the following resolution had been adopted by the Federal Committee on July 14, 1913:

> That the Federal Government be requested to advertise in the daily and medical press all vacant medical appointments and state the salaries to be paid.
>
> That it be a recommendation to the Branches to authorize the Committee to make a similar request to the respective State Governments.

The Committee resolved to reaffirm its decision.

Medical Officers of the Defence Department.

The Honorary Secretary read a communication from the Victorian Branch requesting the Federal Committee to take action in obtaining better rates of pay for medical officers in the Defence Department. Inquiries had been made concerning the practice obtaining in the second military district. It appeared that medical officers attending the personnel at forts and elsewhere were paid the ordinary friendly society lodge rates of twenty-eight shillings per individual. The duties of the medical officer embraced a fortnightly visit to the barracks, control of the sanitary arrangements and attendance on the men. After further information had been elicited it was resolved on the motion of Dr. R. H. Fetherston, seconded by Dr. J. Adam Dick:

> That the Federal Committee suggests that medical officers of the Defence Department be paid not less than the friendly society lodge rates with extra allowances for administrative or similar duties.

Widows and Orphans of Deceased Soldiers.

The attention of the Committee was directed by the Victorian Branch to difficulties that had arisen in connexion with the arrangements made by the Repatriation Department with the friendly societies in regard to the medical attendance on the widows and orphans of soldiers whose deaths had been due to war service and on the widowed mothers of such deceased unmarried soldiers. The Committee was reminded that the Branches had recommended medical officers of friendly society lodges to accept the Repatriation Department beneficiaries on their lists for attendances. The medical officers, however, understood that they were not compelled to do so. It was reported that in Victoria the mileage rate payable under the agreement had not been sanctioned by the Repatriation Department. It was determined to bring the matter before the Repatriation Department, to remind the Department of the conditions set out in the reply of the Department to the Honorary Secretary's letter of November 22, 1923, and to ask that the Department should hold itself responsible not only for the regular contributions, but also for the incidental expenses determined by the agreement. The motion was carried.

The Fatalities at Bundaberg.

A letter was read from the Queensland Branch requesting the Federal Committee to ascertain whether effect would be given to the recommendations contained in the

report of the Royal Commission to inquire into and report upon the deaths and illnesses associated with toxin-antitoxin preparation at Bundaberg.

The Honorary Secretary submitted the following extract from a statement made by the Honourable Sir Neville Howse, Minister of Health, immediately after the issue of the report of the Royal Commission:

> The Minister would like to give the House his personal assurance that he would take the first opportunity of making a personal examination of the whole of the scientific aspects of the Report, but, as the findings of the Commission indicated certain steps which could be taken at once, he would see that those steps were put in hand immediately.

Federal Health Council.

A letter was read from the Queensland Branch requesting that the Federal Committee inquire from the Federal Health Council what steps were being taken to insure that the State representatives of the Federal Health Council promulgated the information they acquired to the medical practitioners practising in the several States. As a result of inquiries it had been ascertained that the Federal Health Council met regularly once a year. The proceedings were published. The representatives of the health departments of the several States returned to their States and no doubt reported fully to their Ministers. It would be a matter for the Ministers to determine what should be done with the information.

Handbook for Recently Qualified Medical Practitioners.

A suggestion was put forward by the Queensland Branch that a handbook for recently qualified medical practitioners should be compiled and issued by the Federal Committee. The Honorary Secretary reminded the members that this matter had been considered by the Federal Committee in February, 1924, and that it had been referred to the Directors of the Australasian Medical Publishing Company, Limited, for their consideration. The Directors had come to the conclusion that as a considerable amount of information on ethical matters and on subjects relative to the practice of medicine was published in the Education Number of THE MEDICAL JOURNAL OF AUSTRALIA, it was doubtful whether the expense that would be entailed in a separate publication, would be justified. Attempts had been made to collect all the information needed for a book of this character by the Honorary Secretary of the Committee for all the Branches and independently by the Department of Public Health of New South Wales for the State of New South Wales. The Honorary Secretary also referred to a book which had been issued by *The Lancet* entitled: "The Conduct of Medical Practice." He thought that the information contained in the publication of the British Medical Association "Handbook for Recently Qualified Medical Practitioners" and in "The Conduct of Medical Practice" covered practically all that was required in Australia. He moved:

> That the Queensland Branch be advised that it is thought that the publication of a handbook for recently qualified medical practitioners in Australia would be too costly.

The motion was seconded by Dr. E. Sandford Jackson and was carried.

Examination for the Fellowship of the Royal College of Surgeons.

At the suggestion of the Queensland Branch the several Branches of the British Medical Association in Australia had been asked to consider the advisability for the holding of the primary examination of the fellowship of the Royal College of Surgeons of England in Australia. It was reported that the College of Surgeons of Australasia had discussed this matter with the Royal College of Surgeons of England. The conditions under which the primary examination was being held in Canada would apply to Australia and New Zealand. This entailed the sending of examiners from England to Australia. The cost of the undertaking would necessarily be high and would have to be defrayed out of the fees charged for the examination.

The members of the Federal Committee expressed the opinion that the majority of Australian graduates would prefer to travel to England for this purpose. On the motion of Dr. F. L. Davies, seconded by Dr. E. Sandford Jackson, it was resolved:

> That the Federal Committee approach the College of Surgeons of Australasia in order to ascertain what arrangements can be made for holding the primary examination of the fellowship of the Royal College of Surgeons of England in Australia.

Name Plates.

A letter read from the Queensland Branch asking for a ruling by the Federal Committee in connexion with the use of non-registerable qualifications on name plates. It was moved by Dr. Gregory Sprott and seconded by Sir Henry Newland:

> That it is unethical to put an unregisterable qualification on a name plate.

After the difficulties arising from the differences in the different States in regard to registerable qualifications had been discussed, Dr. R. H. Fetherston moved and Dr. F. L. Davies seconded that the word "unethical" be deleted and the word "undesirable" substituted. The amendment was lost.

Dr. R. H. Todd moved a further amendment:

> That the view taken by the Federal Committee be that no degree or diploma or any designation indicating a qualification should be used on a name plate.

The amendment was lost.

The original motion was then put to the meeting and was also lost.

Dr. W. H. Crago.

Dr. R. H. Fetherston called attention to the fact that Dr. W. H. Crago, the Honorary Auditor of the Committee, had performed a vast amount of valuable work for the Federal Committee, for the New South Wales Branch and for the medical profession. He thought that the Federal Committee should mark its appreciation of this work and he begged leave to move that the gold medal of the British Medical Association in Australia be presented to Dr. Crago. The motion was seconded by Sir Henry Newland and was carried by acclamation.

Votes of Thanks.

A cordial vote of thanks was accorded to the Council of the New South Wales Branch for their hospitality in entertaining the members at dinner and for its kindness in providing accommodation for the meeting.

A hearty vote of thanks was accorded to Dr. W. N Robertson for the admirable manner in which he had conducted the meeting and for the expedition which he had exhibited.

Date and Place of Next Meeting.

It was decided that the date and place of the next meeting be left in the hands of the Chairman.

Medical Societies.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING OF THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA was held at the University of Adelaide on July 6, 1928.

Manganese Deficiency Disease.

MR. GEOFFREY SAMUEL read a paper on manganese deficiency disease in oats at Mount Gambier. This had previously been investigated by the Agricultural Department and was suggested as a topic of research to the Waite Institute. A Scandinavian paper on a similar disease indicated that it yielded to treatment of the soil with manganese salts. On applying the same method to the

Mount Gambier case a cure was effected. Analysis of the soil, however, showed that the soil was relatively rich in manganese. It was also found that when the soil was sterilized by heat, the disease was cured and for some time this seemed to support the bacterial origin of the condition. Water culture methods, however, had been used to investigate the importance of traces of manganese. Special precautions had been taken in purifying the salts used, as it was found that even the chemicals of the highest commercial purity contained sufficient manganese to prevent the onset of the disease.

The pots in which the cultures were grown, were paraffined inside, as it was found that the glazing contained sufficient manganese to prevent the condition, one part of manganese in 15,000,000 being sufficient. Complete absence of manganese definitely produced the same pathological phenomena as were seen in the field. The questions arose as to why manganese in Mount Gambier soil was unavailable for plants and what was its function in the plant.

The manganese content of the soil at Mount Gambier was the same as that at Urrbrae where there was no such deficiency disease. At Mount Gambier it was found that the disease occurred in strips of land adjoining the roads and as the latter were limestone, it was considered that the limestone dust by raising the alkalinity of the soil had rendered the manganese unavailable. The pot experiments, however, though indicating a certain correlation with this possibility, did not support it as being the sole cause of the trouble. If the soil was treated with acid and subsequently made alkaline, the disease did not occur and the effect of autoclaving was due to an increase in the availability of the manganese caused by the elevation of temperature.

It was found that the manganese contained in soils treated in this manner was elevated when measurements were based upon washing by percolation or with diluted acid. German workers considered that calcareous soil rendered the manganese unavailable. The Dutch, on the other hand, considered that organic matter was the cause of the phenomenon.

The experiments so far carried out by Mr. Samuel did not substantiate either claim. The effect of autoclaving certainly suggested the destruction of an organic compound of manganese. Mr. Samuel then went on to refer to the importance of manganese in the soil which had been noted as early as 1904. Bertrand in his investigations on laccase had in that connexion considered the manganese salt to be present as a coferment. It was finally concluded, however, to be part of the enzyme itself. The actual function of the manganese was still undecided and in this particular connexion it was complicated by the fact that at Mount Gambier the manganese was not necessary for other serials except oats.

In answer to a question, Mr. Samuel stated that manganese was more concentrated in the leaves.

PROFESSOR J. A. PRESCOTT stated that in the Scandinavian conditions the calcium ion was considered the important limiting factor by Arrhenius.

PROFESSOR T. B. ROBERTSON considered that manganese was important for the nucleus itself.

Special Correspondence.

LONDON LETTER.

BY OUR SPECIAL CORRESPONDENT.

Swiney Prize: Medical Jurisprudence.

IT is announced by the Council of the Royal Society of Arts that the next award of the Swiney Prize on medical jurisprudence will be made in January, 1929.

Dr. Swiney died in 1844; it is thus the eighty-fifth anniversary of his death and he left £5,000 in consols to the Society for the purpose that on every fifth anniversary of his death a prize should be presented to the author of the best published work in jurisprudence. The prize is a cup, value £100, and money is awarded to the same amount.

The Royal Society of Arts and the Royal College of Physicians make the award jointly and the prize is offered alternately for medical and general jurisprudence. On the last occasion (1924) the award was for a work on general jurisprudence; it will therefore this year be for a work on medical jurisprudence.

Any person desiring to submit a work in the competition should do so by letter, addressing it to the Secretary of the Society, John Street, Adelphi, London, W.C.2, not later than November 30, 1928.

A New Diploma.

In connexion with maternal mortality it is of great interest to learn that the Society of Apothecaries of London (Apothecaries Hall, Water Lane, E.C.4) has just established a diploma called the "Mastery of Midwifery," the regulations for which have just been published. The first examination for the new diploma will take place in the autumn and from a study of the regulations it is obvious that the standard of theoretical knowledge and of practical proficiency demanded will be a very high one. Candidates must, of course, be fully qualified; in addition they must have held a resident post for six months in a recognized obstetric hospital. Besides this, they must also have attended a recognized ante-natal clinic and an infant welfare centre, the period of attendance in each case being not less than three months. The examination will consist of written papers, a clinical test and an oral examination.

Cancer Clinic for Women.

The Cancer Research Committee of the Medical Women's Federation recently held a meeting at which was discussed the establishment of a clinic solely for the treatment by radium of women suffering from cancer. It was gratifying to hear that a sum of £1,500 had already been received, the subscriptions (chiefly from women) varying from 2s. 6d. to £1,000 and coming from all parts of the country. It was stated that in 1925 27,290 women had died of cancer in England and Wales and it was pointed out that, although cancer was not restricted to one sex, medical women had special opportunities for studying the disease which was most common in women. The fact that the success of treatment depends on getting the patients as early as possible was again emphasized. As excellent results with radium had been obtained at Continental clinics the Committee urged the research officers to make a study of the methods used so that the most successful ones might be adopted over here. In the first instance only those patients whose condition was too advanced for operation, were treated by radium and the results had been so astonishingly good that it was being more frequently suggested that patients in the early stages should use radium rather than have operative treatment. There was no doubt from the evidence obtainable that cancer could be cured by radium. The Committee stated that they had a loan of £4,000 worth of radium and all they needed was a real opportunity to use it, the ideal, of course, being a cancer hospital of their own.

Voluntary Hospitals: Middle Class Patients.

The original intention for which the voluntary hospitals were founded was the care and treatment of the sick "poor" and subscriptions were asked and obtained from the public on the understanding that only "poor" people (that is, those entirely unable to pay fees) were treated. This definition now hardly applies in the same way, as since the establishment and growth of the Poor Law services destitute people are treated in the Poor Law infirmaries, which are under the control of the local Board of Guardians and are paid for by rates.

The voluntary hospitals now tend to deal more particularly with the class of small wage-earners—manual labourers, shop assistants, the lower grades of clerical workers *et cetera*—who are in most cases capable of paying a few shillings towards the cost of treatment. This class might be more accurately defined as those workers who earn enough to live on and bring up families (it being borne in mind that "bringing up" does not include

education and other services which are free), but not enough to bear more than a tiny proportion of the expenses of medical treatment. Towards the cost of possible illness it should be noted that workers earning less than £250 *per annum* compulsorily pay a weekly contribution from their salaries to the National Health Insurance Fund. There are also voluntary savings associations or contributory schemes in connexion with hospital benefits, in all cases with an income of limit which is usually four pounds per week for a single person, five pounds for man and wife without dependent children and six pounds for man and wife with dependent children.

At the other end of the scale comes the "wealthy" class which is quite capable of looking after itself.

In between these two extremes, represented on the one hand by the very low income limits (£200 or £300) allowed by the hospitals and on the other by the wealthy, comes a huge and unfortunate middle class which is ineligible for hospital and medical treatment (whether entirely free or partially paid for by National Insurance or contributory schemes) and is unable to pay nursing home charges. This class is considered and rightly to be the backbone of the community; its members, being rate-payers, contribute to the cost of succouring the destitute poor, but for its own problem of how to cope with illness and its attendant expenses there is at present no satisfactory solution. By means of paying wards in some of the hospitals the fringe of the trouble has been touched, but the number of patients that can be dealt with in this way, is negligible when considered in relation to the size of the "middle class."

Correspondence.

INCIDENCE OF VENEREAL DISEASE IN THE ARMY AND IN THE CIVIL POPULATION.

SIR: The report for 1921 showed a ratio of 74 venereal disease cases per 1,000 in the British Army and this fell to 47·28 in 1924. In the "V.D. information" for May, 1928, issued by the Surgeon-General of the United States it is stated that the ratio was 44·4 per 1,000 for the whole army at home and abroad in 1925. This has fallen in 1926 to 41·4 per 1,000, *id est* almost half the rate for 1921. In the same issue a reference is made to the hopes of those in New York State who have been conducting an intensive anti-venereal disease campaign and have been expecting a decrease in the number of cases of syphilis in civil life.

The following table gives the rate for syphilis in the State excluding New York City.

	Cases per 100,000 of population.
1919	191
1920	194
1921	198
1922	209
1923	230
1924	228
1925	270
1926	252
1927	248

The report concludes: "The statistics seem to indicate the possibility of the crest having been reached. However, the decline in the past two years has been so small and the time so short that it can be considered only an indication. Nevertheless, it is a hopeful sign." How much longer are we to wait until the medical profession takes up the campaign against venereal diseases seriously. In my judgement the problem rests in the hands of the backbone of the medical profession, *id est* the general practitioner.

Yours, etc.,

JAMES W. BARRETT.

[The figures quoted by Sir James Barrett, it is assumed, are supposed to reveal a reduction of venereal disease in the British Army and the reverse in the rural population of an American State. It is strange that figures relative to the incidence of a disease among British soldiers should be ascertained from a source outside the Empire. Even if they are reliable, they prove very little. It has long been recognized that when men are placed under discipline, the incidence of gonorrhœa and to a less extent of syphilis diminishes. In the British Army the reduction of infections is probably caused to some extent by the facilities provided for prophylactic measures and to some extent to the fact that men who acquire infections, are subjected promptly to treatment. The figures given in regard to syphilitic infections in the New York State, excluding the great metropolitan area of New York City, are valueless. There is no evidence that they represent the incidence of syphilitic infections. But even if they do correspond more or less accurately to the rate of incidence, there is nothing to prove that the variations have been affected by the intensive campaign. Sir James Barrett has failed to support his contention that educational measures have any effect on the incidence of these diseases. If the figures given mean anything, they mean that these diseases may be checked to some extent when stern measures, disciplinary control and a knowledge of the amount of infection enable those in authority to reduce the sources of infection in the males. Preaching and telling the people all about sexual physiology and sexual diseases have taken no part in the reduction of infection among soldiers.—EDITOR.]

USE OF "INSULIN."

SIR: In your issue of August 11 in reporting the meeting held at Mooroopna, I am made to say that I had recently treated a patient with "Insulin" tablets given by the mouth and that they were as effective as the liquid preparation given hypodermically.

I only wish this were true, but unfortunately it is not. I told the members that I had found tablet "Insulin" quite effective; but, of course, it has to be given hypodermically. It has a distinct advantage for patients living in the tropics, as did my patient.

Yours, etc.,

JOHN F. WILKINSON.

55, Collins Street, Melbourne.
August 13, 1928.

DENGUE FEVER.

SIR: I much appreciate Dr. Cilento's comment in your edition of August 18 on the report of my case of dengue fever in your issue of August 4, 1928. Through an oversight on my part the differential diagnosis was not discussed in my report. Endemic typhus fever was definitely excluded, the Weil-Felix test being negative (Board of Health, Sydney). Other conditions that were ruled out were influenza, acute epidemic cerebro-spinal meningitis (I saw many cases during 1915-1916 epidemics, ushered in as this case was, with a similar eruption especially on the buccal and pharyngeal mucous membrane), acute rheumatism, measles, scarlet fever, "fourth" disease, typhoid fever, drug eruptions, food poisonings *et cetera*.

Having seen a fair amount of dengue fever, especially when I was in practice in the Wyong district, about sixty miles north of Sydney, and having seen it there as late as June and having excluded the above conditions, I felt my diagnosis of dengue fever was justified and being so unusual, thought worth reporting.

Yours, etc.,

REG. E. NOWLAND.

Balmain.
August 18, 1928.

FAULTS IN THE UTERINE POWERS.

SIR: I was extremely interested in Dr. A. J. Gibson's article on the above subject in THE MEDICAL JOURNAL OF AUSTRALIA of August 11, 1928. In the first place I have

very grateful memories for Dr. Gibson of my first night in charge of the Women's Hospital, Crown Street, a number of years ago, being sent there whilst a resident at the Royal Prince Alfred Hospital, when a case necessitated the application of forceps. One of the honoraries instructed me to "put them on," which I did, but could not move the head, so I got Dr. Gibson to come along. He supplied the power that was lacking in my comparatively feeble arm and delivered a hefty young fellow of about thirteen or fourteen pounds. What interested me mostly, however, was "The Innervation of the Uterus."

Dr. Gibson's description does not accurately coincide with any authority I have studied nor with my own findings spread over the last year or so. He describes "the great uterine plexus" situated above the promontory of the sacrum near the bifurcation of the aorta. There certainly is a huge plexus at that situation, but it is by no means confined to the innervation of the uterus, hence I think the name is a misnomer and confusing.

Most authorities—Hovelacque *et cetera*—refer to this plexus as the superior hypogastric plexus and in my work I have found the following connexions:

1. With the vertebral chain or gangliated cord.
2. The inferior mesenteric plexus, ? the aortic plexus.
3. The inferior hypogastric plexus, situated on the sides of the rectum at about the level of the junction of the cervix and *corpus uteri* and from which the main nerve supply passes to the uterus.
4. A fair number of nerves from this plexus (superior hypogastric) pass to the posterior aspect of the pelvic colon and rectum and one or two may reach the "so-called ganglionic mass" at the junction of the cervix and *corpus uteri*.

I use this term "so-called ganglionic mass" advisedly, because the nature of this structure is debatable. Many authorities, Snowbeck, Dahl *et cetera*, describe it as a mass of connective tissue in which are placed a few small ganglia the size of a pin's head and which receive medullated and non-medullated nerve fibres. I persume this is the structure referred to by Dr. Gibson as "the great cervical ganglion." If this is admitted, an easier explanation of the enlargement described by Dr. Gibson during pregnancy is forthcoming than if it were a true nervous ganglion.

In the present state of our knowledge, anatomical physiological *et cetera*, I do not think Dr. Gibson's statement that "the presentation stimulates the great cervical ganglion" can be admitted unchallenged.

As I intend publishing the results of my researches in due course, I shall not go into further detail just now and hope my remarks will not be taken as destructive criticism of Dr. Gibson's interesting and useful paper.

Yours, etc.,

REG. E. NOWLAND.

Department of Anatomy,
Sydney University.
August 14, 1928.

A SIMPLE DIABETIC FORMULA.

SIR: Below are some simple diabetic formulæ which, as far as I can find out, are quite new. I hope that they are also useful. My aim has been to provide formulæ not only as simple as Campbell's or Wilder's, but formulæ which will also automatically give any desired $^{FA}/_{G}$ ratio. This can be done by employing a fixed $^{P}/_{M}$ ratio (where P is the number of grammes protein and M is the total number of calories in the diet, whether calculated from weight, by Du Bois's chart or by any other method), for example $\frac{P}{M} = \frac{1}{25}$ or $\frac{1}{30}$ and this I have found satisfactory.

Thus let $\frac{P}{M} = \frac{1}{30}$

and $\frac{FA}{G} = \frac{0{\cdot}44P + 0{\cdot}9F}{C + 0{\cdot}58P + 0{\cdot}1F} = X$

and a basic formula can be readily drawn up in which by simple substitution for varying values of X we can draw up tables like the following. Moreover, the same can be done for any required $^{P}/_{M}$ ratio.

$\frac{FA}{G}$	Carbohydrate.	Protein.	Fat.
1·0	5·3% of M	$\frac{M}{30}$	7·3% of M
1·1	4·8% of M	$\frac{M}{30}$	7·5% of M
1·2	4·2% of M	$\frac{M}{30}$	7·8% of M
1·3	3·9% of M	$\frac{M}{30}$	7·9% of M
1·4	3·5% of M	$\frac{M}{30}$	8·1% of M
1·5	3·2% of M	$\frac{M}{30}$	8·2% of M

Example:

Desired $\frac{P}{M} = \frac{1}{30}$

Desired $\frac{FA}{G} = 1{\cdot}5$

Calories 1,600.

$C = 3{\cdot}2 \times 16 = 51$ grammes.

$P = \frac{160}{3} = 53$ grammes.

$F = 8{\cdot}2 \times 16 = 131$ grammes.

The figures are not exact, but sufficiently close for all practical purposes.

Yours, etc.,

C. C. MCKELLAR,
Resident Medical Officer,
Royal Alexandra Hospital for Children.

August 8, 1928.

Congresses.

INTERNATIONAL PHYSIOLOGICAL CONGRESS.

THE thirteenth International Physiological Congress will meet at the Medical School of Harvard University, Boston, Massachusetts, from August 19 to 23, 1929. The Federation of American Societies for Experimental Biology which comprises the American Physiological Society, The American Society of Biological Chemists, The American Society for Experimental Pathology and The American Society for Pharmacology and Experimental Therapeutics, will serve as hosts to the Congress and Professor William H. Howell, of Johns Hopkins University, will be the president. The arrangements for the Congress are in the care of Professor Walter B. Cannon of the Harvard Medical School, who is Chairman of the Congress Bureau, and Professor Edwin J. Cohn and Professor Alfred C. Redfield who are the Secretaries.

Honours.

HIS EXCELLENCY THE GOVERNOR OF NEW SOUTH WALES presented to Dr. H. R. G. Poate, the Acting Assistant Commissioner in charge of the New South Wales District of the Saint John Ambulance Brigade, the insignia of a serving Brother of the Order of Saint John. The ceremony took place on August 9, 1928.

Proceedings of the Australian Medical Boards.

NEW SOUTH WALES.

THE undermentioned have been registered under the provisions of *The Medical Act* 1912 *and* 1915, of New South Wales, as duly qualified medical practitioners:

Brown, John Noel, M.B., B.S., 1918 (Univ. Melbourne), Albury.

Day, Ellen Jemima Mary, M.B., B.S., 1926 (Univ. Melbourne), Albury District Hospital.

McKay, Donald, M.B., Ch.B., 1915 (Univ. New Zealand), c.o. L. Levick, Esquire, Agincourt Road, Eastwood.

Raysmith, Frank Walker, L.R.C.P. (Edinburgh), 1928, L.R.C.S. (Edinburgh), 1928, L.R.F.P.S. (Glasgow), 1928, Kenmore Mental Hospital, Goulburn.

Street, Raymond Edward, M.B., B.S., 1924 (Univ. Melbourne), Prince of Wales Hospital, Randwick.

Swanton, Cedric Howell, M.B., B.S., 1924 (Univ. Melbourne), F.R.C.S. (Edinburgh), 1926, Chertsey Street, Merrylands.

Books Received.

A TEXT-BOOK OF BIOLOGIC ASSAYS, by Paul S. Pittenger, Ph.G., Ph.C., Ph.M., Phar.D.; 1928. London: P. Blakiston's Son and Company. Post 8vo., pp. 396, with illustrations.

THE PRESCRIBING OF SPECTACLES, by Archibald Stanley Percival, M.A., M.B., B.C.(Cantab.); 1928. Bristol: John Wright and Sons, Limited. Crown 8vo., pp. 239. Price: 15s. net.

AIDS TO DISPENSING, by A. O. Bentley, Ph.C.; 1928. London: Baillière, Tindall and Cox. Foolscap 8vo., pp. 200. Price: 3s. 6d. net.

Diary for the Month.

AUG. 28.—Illawarra Suburbs Medical Association, New South Wales.
AUG. 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
AUG. 30.—New South Wales Branch, B.M.A.: Branch.
AUG. 30.—South Australian Branch, B.M.A.: Branch.
SEPT. 4.—Tasmanian Branch, B.M.A.: Council.
SEPT. 5.—Victorian Branch, B.M.A.: Branch.
SEPT. 5.—Western Australian Branch, B.M.A.: Council.
SEPT. 5.—South Sydney Medical Association, New South Wales.
SEPT. 6.—South Australian Branch, B.M.A.: Council.
SEPT. 7.—Queensland Branch, B.M.A.: Branch.

Medical Appointments.

Dr. Louis Joel (B.M.A.) has been appointed Public Vaccinator at Newport, Victoria.

* * * *

Dr. Daniel Patrick O'Brien (B.M.A.) has been appointed Medical Officer, Westwood Sanatorium, *viâ* Rockhampton, Queensland.

* * * *

Dr. John Albert Emmett (B.M.A.) has been appointed Government Medical Officer at Ayr and a Health Officer for the purposes of *The Health Acts* 1900 *to* 1922, of Queensland.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii.

COMMONWEALTH OF AUSTRALIA: Medical Officer.
LAUNCESTON PUBLIC HOSPITAL: Junior Resident Medical Officer (Male).
ROYAL NORTH SHORE HOSPITAL OF SYDNEY: Honorary Clinical Assistant, Orthopædic Section.
SWAN HILL DISTRICT HOSPITAL: Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. Marrickville United Friendly Societies' Dispensary. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to position at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor." THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

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